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THE
LARYNGOSCOPE.

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A REVIEW OF THE AVAILABLE LITERATURE ON THE PHARYNX AND PHARYNGEAL SURGERY FOR 1950.*

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ANATOMY.

The presence of a canal extending from the depth of the sella turcica to a point just behind the rostrum of the sphenoid has been reported many times and has been named the craniopharyngeal canal. Its presence is frequently noted in the higher apes. It is not uncommon in infancy and is rather rare in adults. Heretofore it has been assumed to be a persistent opening from the growth of Rathke's pouch. The investigation of Arey[1] disproves that origin and states that the craniopharyngeal canal is a channel formed during osteogenesis by the growth of blood vessels into the bone marrow in this region in the fetus.

Adhesions of the nasopharynx consisting of string-like fibrous bands to actual sheets of scar tissue are commonly seen in routine examination of this area. Lion[2] describes a peculiar type of headache located around and behind the eye, which he attributes to inflammation and retention of material in the nasopharynx behind these folds. Various folds and adhesions are classified by the direction they take. Lion be-

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lieves these adhesions are caused by inflammatory change in this area and to a lesser degree by improperly performed adenoidectomies, and that only a small percentage are congenital. Treatment consists of early and properly performed adenoidectomy as a preventive measure and simple clipping of the fibrous bands once they are formed.

In a symposium on irradiation of lymphoid tissue in the nasopharynx, Schenck[3] discusses the anatomy, physiology and pathology of nasopharyngeal lymphoid tissue. He reminds us that at the chief portals of entry to the body there is urgent need of a mechanism of removing and attenuating organisms and of developing immunity against them. It is here that lymph cells in large numbers are found. Some of the masses of lymph cells are so large they have been dignified by the term "tonsil." The innumerable smaller collections of lymph cells, commonly referred to as "follicles" or "nodules," are scattered throughout the pharynx and adjoining structures. These lymph structures are an integral part of a universally distributed system made up of lymph channels and vessels, and of accumulations of lymphocytes called lymph nodes, of which the body contains from 500 to 1,000, representing 1 per cent of total body weight. Lymph tissue, whenever found, is composed of two chief constituents: the supporting connective tissue reticulum and the lymph cells contained within the meshes of the reticulum. Lymph nodules and lymph nodes are discussed in a most excellent manner. Lymph tissue and lymphocytes play an important rôle in resistance and are probably the site of antibody formation. Because of the clinical importance of excessive growth of lymph tissue in the nasopharynx, Schenck thinks it advisable that consideration be given the factors which govern the growth of lymph tissue in general. He enumerates the most important as diet, endocrine secretions, stimuli other than hormones, infection and heredity. Each of these is discussed at length. This article constitutes probably the most concise description of lymph nodules, nodes and tissue structure of the nasopharynx available and describes in an interesting manner the complicated function of the lymphatic system.

DISEASES.

Postnasal discharge is a common and troublesome complaint, which is often entirely physiologic. Tremble[4] believes that low grade sinusitis involving the antra, ethmoids and sphenoids, chronic hypertrophic rhinitis, especially of the inferior turbinates, and lymphoid hyperplasia of the nasopharynx, are the commonest causes of this condition. Numerous other causes including endocrine imbalance and abuse of nose drops are listed. The physiology of the nose and sinuses is reviewed.

Treatment of the sinusitis consists in irrigations and displacement therapy. Hypertrophic inferior turbinates may be reduced in size by submucosal electrocoagulation with twin electrodes, or by surface coagulation with fuming nitric acid. Use of fuming nitric acid to enlarge the nasofrontal duct is also advocated. For other cases in which definite causes can be determined, specific therapy is indicated.

This article seems to minimize the part played by allergy in the average patient complaining of a postnasal discharge.

A case of actinomycosis of the tongue and pharynx manifested by superficial excrescences filling crypts in the involved areas and successfully treated with chloromycetin is presented by Wilkerson and Cayce.[5]

At the Fifty-ninth Meeting of the Scottish Otological and Laryngological Society, Howie[6] reported two cases of amyloidosis of the pharynx and larynx. One of the patients had pulmonary tuberculosis. A classification of amyloid disease of the larynx, as published by New, is quoted.

Wilson[7] presents a case of myoclonus of the palate, pharynx and larynx in a woman 61 years old. He presumed that it was due to a petechial hemorrhage near the IIIrd nerve nucleus.

Williams[8] directs attention again to the oral and pharyngeal complications that might arise following chloromycetin therapy. Of 200 patients receiving chloromycetin therapy, observed by Williams, oral and pharyngeal complications, con-

sisting of glossitis, black tongue, stomatitis, pharyngitis and monilia albicans infection, developed in 12, or 6 per cent. Bitterness, dry mouth and soreness of the mouth usually appear four to six days after administration of the drug. The more severe complications were seen in elderly or debilitated patients. The lesions usually subside in the week following discontinuance of the drug. Administration of vitamins did not alter the complications; patients receiving additional antibiotics had more severe complications. The opinion is expressed that the monilia is held in check by the bacterial flora of the mouth and pharynx and this balance is upset by the antibiotic.

Webb[9] reports a case of Guillain-Barré syndrome with respiratory and pharyngeal paralysis. With the aid of a respirator and tracheotomy the patient recovered and was able to walk after six weeks. This condition is a polyneuritis of unknown etiology with a good prognosis. Of 115 cases in the literature, 25 patients died, practically all of them of respiratory paralysis. Early tracheotomy and early use of a respirator would seem to prevent most of these deaths.

Smith[10] reports a case of tuberculous retropharyngeal abscess in a woman 35 years old, in whom cure was effected by surgical treatment and administration of streptomycin. The unusual feature of the case was the lack of involvement of the cervical vertebrae, as is usually the case in tuberculous abscesses in the pharynx.

Bryant[11] reports a case of retropharyngeal abscess in a boy six years old, which was thought to be secondary to infection in a regrowth of adenoid tissue. The abscess was successfully incised and drained, and two months later a mass of adenoid tissue was removed.

Schenck[12] presents a practical textbook type of discussion of several acute infectious conditions of the pharynx, and although it is fairly elemental, there are a few good points emphasized under each heading. Septic sore throat is due to hemolytic streptococcus; warm saline irrigations, local applications of tincture of benzoin and penicillin parenterally are

recommended. Acute lacunar tonsillitis may be treated by applications of 25 to 50 per cent silver nitrate directly to the tonsils, using a small amount of the solution, warm saline irrigations and parenteral penicillin. It is important to study the blood picture carefully in patients with Vincent's angina to rule out leucemia. Treatment of Vincent's angina consists of applications of sodium perborate paste, parenteral and local administration of penicillin, and in some cases intramuscular injection of sodium cacodylate. When diphtheria is suspected, culture on Loeffler's medium should be done. Toxoid injections are imperative and penicillin is useful in combatting secondary infection. Peritonsillar abscess may be aborted by penicillin or sulfonamide therapy, but once fluctuation occurs, incision and drainage must be done. Acute pharyngitis may be treated by local application of compound tincture of benzoin and saline irrigations; it often heralds a systemic disease. Thornwaldt's disease, due to infection and cyst formation of a pharyngeal bursa, is treated by surgical removal of the bursa. Retropharyngeal abscess occurs almost entirely in children and is due to suppuration of the retropharyngeal lymph nodes; incision and drainage are employed once fluctuation occurs. Penicillin parenterally will usually prevent this serious condition.

Asherson[13] presents some cases with excellent radiographs to draw attention to the syndrome of achalasia of the cricopharyngeus. This condition, meaning failure of relaxation, may occur at the cardia or lower end of the esophagus, or at the upper sphincter of the esophagus, the cricopharyngeal sphincter. In the latter case it results from failure of this sphincter to relax when a bolus of food approaches it. Achalasia of the cricopharyngeus presents a typical radiological picture; on lateral Roentgenogram the hypopharynx is elongated and the valleculae are widened. Barium may take over five minutes to pass. This condition may be produced by pharyngeal paralysis such as caused by poliomyelitis, thyrotoxicosis, myopathy, unilateral recurrent laryngeal nerve paralysis, after partial pharyngectomy and in bulbar paralysis.

The condition is manifested clinically only by slight dysphagia, and on examination pooling of mucus is seen in the pyriform fossa. Esophagoscopy is indicated to rule out postcricoid neoplasm or a pharyngoesophageal diverticulum. Other conditions to be considered in the differential diagnosis of achalasia of the cricopharyngeal sphincter are Plummer-Vinson syndrome, foreign body of the esophagus, idiopathic dysphagia in the female and cricopharyngeal spasm produced by overdistension.

Crowe and associates[14] present a brief review of the highlights of the literature on otorhinolaryngology for the period of 1938-1948. The use of antibiotics is discussed; a small section is devoted to carcinoma of the sinuses, nasopharynx, mouth, hypopharynx, larynx and esophagus. The importance of chronic irritation as a cause of cancer of the mouth and pharynx as presented by Hayes Martin is mentioned.

DIAGNOSIS.

Lindsay and Perlman[15] present an excellent discussion of tests of chronic Eustachian tube obstruction. From a practical standpoint they divide the tests into three groups according to the age of the patient. In group one is placed the preschool child. The earliest practical indication of chronic tubal obstruction in this group is the presence of fluid in the middle ear. When distortions and scarrings of the drum are present, middle ear changes of irreversible nature have usually occurred. Early diagnosis depends upon the ability of the examiner to recognize the signs of a middle ear completely filled with fluid as well as one showing fluid lines. In group two are placed young school children whose hearing tests are limited in value. Otoscopic appearance is the chief indication of chronic tubal obstruction. Toynbee's maneuver, if positive, indicates grossly adequate function. In the third group are placed older children and adults.

Tubal function is determined by otoscopic examination and specialized tests. Detailed description of method employed in the detection of tubal function should be of utmost value to the student of otology who is not versed in these procedures.

DIVERTICULA.

The majority of diverticula originate between the oblique fibres of the inferior constrictor and the cricopharyngeus and are found on the left side of the neck. They consist of an outer fibrous and inner mucosal layer. The etiology is in dispute, but because of the occurrence of six cases in one family, Dunhill[16] favors a congenital defect rather than neuromuscular dysfunction as the cause. The usual symptoms are dysphagia, inability to swallow and regurgitation of food; severe cough may occur. The diagnosis is made from the history and Roentgenographic demonstration of the pouch.

For many years the two-stage procedure was preferred because of the danger of mediastinitis, and diverticulopexy, in which the pouch was elevated and sutured with the sac higher than the neck, has been used, particularly in older patients. More recently removal of the diverticulum in one stage has been widely employed. This is the method advocated by Dunhill for those experienced in neck surgery. The pouch is examined endoscopically only in patients in whom the possibility of carcinomatous invasion is suspected. The technique described is the generally accepted one in this country.

Because he believed that the importance of a pharyngeal diverticulum in the production of pulmonary complications is not commonly recognized, Wilson[17] reports the case of a man 61 years old with a pharyngeal diverticulum which was causing difficulty in swallowing, "chest trouble," and "laryngitis" followed by a persistent cough. After excision of the diverticulum, which was located at the level of the upper border of the manubrium, the patient gained weight, and the cough, though still present, was not as persistent or productive as before operation.

Koch[18] presents the results from six months to 12 years after surgical treatment of pharyngoesophageal diverticula in 10 patients ranging in age from 48 to 82 years. In all cases the diverticulum was removed in one stage, under local anesthesia in nine and intubation narcosis in the other. Complications with pneumonia developed in three cases, resulting in

death in one of these. Follow-up examination revealed absence of symptoms and no evidence of recurrence in all nine patients. Postoperative stricture requiring dilatation did not occur in any of the cases.

In the discussion of the surgical management of pharyngoesophageal diverticula, Vollenweider[19] describes the various stages of development of these pouches and discusses the symptomatology. Attention is directed to persistent cough, excessive secretion and a sensation of fullness in the throat. Dysphagia is largely determined by the size of the sac, its retention and the pressure exerted on the esophagus. Palliative treatment offers no hope, and Vollenweider warns of the danger of perforation from blind introduction of bougies into the esophagus.

Vollenweider advocates the two-stage operation for extirpation of the diverticulum; the second stage being performed two weeks after the first. During the last three years five cases were performed. These are briefly described.

The problem of accurate dissection of a pharyngoesophageal diverticulum may be difficult, and removal of the diverticulum and suture of the esophageal defect in the past has often resulted in a stricture at the operative site. Terry[20] recommends aspiration of the pouch followed by illumination of it with a specially designed light which is left in the pouch during the operation and passage of a large Magill tube into the esophagus preliminary to removal of the pouch. Intratracheal anesthetic is used. This technique has been employed successfully in five cases. Use of the esophagoscope for illuminating the pouch and fixing and dilating the upper part of the esophagus seems to us a much simpler and just as satisfactory procedure.

Negus[21] presents an excellent study of pharyngoesophageal diverticula, which should be read in its original form by all handling these cases. The evolution of the anatomy of the hypopharynx and upper end of the esophagus and its relationship to the larynx is completely presented. Negus gives the following factors as predisposing to the formation of a

pouch: the low position of the larynx to mouth of esophagus in man; the arrangement of the oblique fibres of the inferior constrictor and the cricopharyngeus; the anterior attachment of the esophagus to the larynx; difficulties in coordination between the cricopharyngeus and the pharyngeal force pump and the absence of support at an unprotected spot. Since all other factors are present in everyone, the variable factor of incoordination of relaxation of the sphincter is given as the principal cause of pharyngoesophageal diverticula.

Early small pouches may respond to repeated dilatations of the cricopharyngeus and for technical reasons it is thought best not to operate on the small pouches. Negus prefers one-stage removal of the diverticulum but leaves the wound open to heal by secondary intention. No mention is made of hereditary factors in the etiology of diverticula.

Buckstein and Reich[22] present two cases of lateral pharyngeal diverticula. These sacs extend anterolaterally from the pyriform fossa and are thought to be due to unobliterated third and fourth bronchial pouches. They cause symptoms of dysphagia and regurgitation of undigested food. Diagnosis is made by spot films of the hypopharynx with barium swallow. No mention is made of indirect or direct laryngoscopic examination or of the treatment of this condition.

FOREIGN BODY.

Williams[23] reports the case of a man with chronic schizophrenia, aged 32 years, who swallowed a crucifix, 5 cm. by 3.2 cm., which lodged in the upper part of the esophagus and was succesfully removed under general anesthesia.

Wolfsohn[24] reports the case of a patient who ingested a wire foreign body which was removed from the upper esophagus within 24 hours. Several weeks later, mediastinitis developed, but this responded to antibiotic therapy. Two and one-half months later she complained of a clicking sound on swallowing and of regurgitation. Examination following ingestion of barium revealed a typical pharyngoesophageal diverticulum.

INSTRUMENTS.

Price[25] was prompted to construct an apparatus for aspiration of posterior pharyngeal secretions because of repeated failure to demonstrate eosinophilic cells in routine smears of nasal secretions obtained from sputum smears, smears of freshly swabbed secretions from the anterior nares and smears of nasal discharge or swabbings from the posterior nasopharynx. The apparatus consists of a shortened 20 ml. Fisher pipette of 0.6 cm. bore, a small rubber bulb, and a small length of flexible rubber tubing of 0.7 cm. diameter.

The smear is usually obtained about one-half hour after the patient has awakened in the morning. With two tongue depressors gently depressing the base of the tongue and so placed against the teeth as to prevent sudden closure of the mouth, the aspirator (with hand bulb compressed) is quickly slipped into the throat. Mucus is suctioned from the posterior pharyngeal wall into the pipette barrel, after which the apparatus is promptly withdrawn. Thin smears of the secretions thus obtained are immediately prepared on glass slides and when dry are examined microscopically.

IRRADIATION.

From 1939-1940, 1,365 white school children were examined at the Johns Hopkins University School of Medicine Otological Research Laboratory. A two-year follow-up was reported in 1942. As many of the children as possible were re-examined during 1946-1947. A comparison of the records of these two examinations is presented in a report by Guild.[26] The average interval between examinations was six and one-half years. All children had careful ear, nose and throat examinations including nasopharyngoscopy and audiometric studies. Some were treated for deafness by nasopharyngeal irradiation and others by surgical procedures. The conditions were the same at re-examination as at the original examination.

The largest group, 259 children, had normal hearing at the first examination and received no treatment. They showed a

slight gain in hearing in the low frequencies and a slight loss in the high frequencies. A large number of this group had abnormal tubal orifices at both examinations.

The ears of 95 children who had impairment for high tones only were treated by nasopharyngeal irradiation. They showed the same gain in low frequencies as the untreated group and in addition a slightly greater loss for the high frequencies. About half of the 24 children with moderate hearing loss for all tones treated by irradiation showed moderate improvement in hearing for all tones.

We do not consider the evidence in this study to be conclusive with regard to the treatment of moderate deafness for all tones by nasopharyngeal irradiation. Perhaps the most useful information gained in this study is the normal change in hearing acuity over a six and one-half-year period in children; it would seem that this study would tend to decrease the indiscriminate use of nasopharyngeal irradiation in children.

Smith and Scharfe[27] present a brief review of the literature on irradiation treatment of deafness. They applied 50 mg. radium sulphate enclosed in a 0.3 mm. monel metal chamber to each side of the nasopharynx for eight and one-half minutes at two to three-week intervals for three treatments. Treatments were given to 50 patients; 28 had Eustachian tube obstruction, seven had mixed deafness and 12 had ears with chronic suppurative otitis media. From a study of their cases they were able to make several generalizations. Irradiation is effective in removing excessive nasopharyngeal lymphoid tissue but should be preceded by surgical removal of an adenoid mass if present. Other causes of Eustachian tube obstruction should be sought before irradiation is employed as it is not without danger. Improvement in hearing should not be expected in patients with nerve deafness or mixed deafness or in those in whom irreversible fibrous changes have occurred in the middle ear.

This article simply confirms the observations of others in the tremendous amount of literature published on this subject in the past few years.

A brief historical review of the use of irradiation in the pharynx and nasopharynx is presented by Lederer,[28] who believes that there is no danger in the use of nasopharyngeal irradiation if standard technique is followed. Best results are obtained in children with conduction deafness due to lymphoid hyperplasia around the Eustachian tube orifices. The importance of careful history taking regarding previous irradiation is stressed.

In the carefully controlled series of treatments with the 50 mg. monel metal nasopharyngeal radium applicator reported by Rubin and associates,[29] the amount of irradiation received by the physician was measured. Average exposure to the fingertips was 50 mr per treatment. Exposure to the remainder of the finger and the body was considerably less. Rubin and associates recommend 600 mr per week, the maximum exposure to the hands and fingers. On this basis, a maximum of 12 treatments per week should be the limit for any one physician.

An excellent set of instructions for personnel handling the radium applicator is given. It would seem well worthwhile to post these instructions in any office where this form of therapy is employed.

A study based on the use of irradiation of the nasopharynx with the standard 50 mg. radium monel applicator in 468 cases is presented by Bilchick and Kolar.[30] The calculated dosage is roughly estimated to be 2,860 r. at 1 mm. in $12\frac{1}{2}$ minutes and is composed of 75 per cent beta and 25 per cent gamma rays. There is a tendency for patients to notice a dry, irritated throat 24 to 48 hours after operation and for preexisting otitis media to flare up on the eleventh to thirteenth day, indicating the height of the erythema. The action of the irradiation is primarily on the lymphoid tissue, but changes in the epithelium of the nasopharynx also occur and here lies the potential danger of this therapy; however, no case of

malignant change or other complication has been reported from the use of the radium applicator. Irradiation in this series was done on patients with conduction deafness, nerve deafness, postnasal discharge and several other conditions. Good results were obtained in cases of conduction deafness, particularly in children. Little if any benefit was obtained in its use for other conditions. Care in handling of the applicator is emphasized. It is advised that large amounts of lymphoid tissue can be removed surgically before irradiation.

Extensive experiments by Braestrup[31] were conducted to determine accurately the dosage of radiation delivered by a 50 mg. radium monel applicator. It was possible to measure both gamma and beta radiation. This was important as the monel applicator emits both; however, beta rays predominate only at the surface. These experiments indicate clearly that dosages commonly used in nasopharyngeal irradiation exceed those applied in any other type of radiotherapy for nonmalignant conditions; however, the high intensity is limited to a small volume of tissue close to the applicator. This explains why so few cases of radiation damage have been mentioned in the literature. This is a highly technical article and should be studied by all interested in dosage penetration of gamma and beta rays.

Lampe[32] warns that use of irradiation to the nasopharynx can conccivably be productive of harmful results in years to come, although following prescribed dosage there should be no danger attending its use. Radiation therapists employing radium have learned, sometimes by bitter experience, that radiation damage may become manifest years after a course of treatment which resulted in no apparent immediate harm. With nasopharyngeal beta irradiation, the potential biologic dangers must be considered both in relation to the physician applying the radium and in regard to the patient. Lampe warns that there is danger in repeating the applications of radium to the nasopharynx which greatly increases the potentiality of inducing severe vascular and connective tissue to the point where the tissue may become vulnerable to the insults of trauma and infection.

In a most interesting article, Day[33] does not deny that good results have followed use of irradiation; however, he does not believe that lymphoid tissue in the vicinity of the tubal orifice is responsible for tubal closure and deafness in the great majority of cases and he believes that irradiation of such tissue will seldom relieve deafness or cause the tubes to open. In spite of glowing reports of irradiation, Day has been greatly disappointed by his experience with this form of therapy. Two-thirds of all patients under his observation had a nasal allergy which had to be controlled before the aural symptoms were entirely relieved. Irradiation gave temporary relief to some of the allergic patients; but the tubes again became closed, and the deafness returned in six to nine months. The second most common cause of tubal obstruction in children, according to Day, is the presence of masses of adenoids or adhesions. It is surprising that only 10 per cent of cases of conductive deafness with tubal closure were corrected by removal of tonsils and adenoids. Day discusses prevention and prophylactic measures which should begin with the baby in its cradle and he believes that many hearing defects are due to improper feeding of babies. Unless the infant's head is elevated at least 30 degrees from the horizontal, it cannot swallow without forcing fluids into the nasopharynx. Day is of the opinion that on occasions the application of radium to the nasopharynx may be productive of harm, particularly if repeated too often. The nasopharynx should be examined carefully before the applicators are inserted, and the use of these applicators should be confined to conductive types of deafness. He describes a suggested experiment to which each user should subject himself so as thoroughly to appreciate the potency of the therapeutic agent. Day's article is replete with interesting data and common sense and should be read by all interested in this phase of therapy.

Bordley[34] emphasizes the fact that infected lymphoid tissue in the nasopharynx has long been recognized as one of the principal factors to recurrent acute and chronic infections of the upper air passages and ears. For years numerous surgical

and medical procedures have been used unsuccessfully to clear up such infections. In 1924, Crowe and Burnam used irradiation successfully in a series of patients suffering from auditory obstruction caused by hypertrophied lymphoid issue in the region of the Eustachian orifices. This method of therapy has been accepted universally as having merit in selected cases. It is effective in treating conductive type hearing impairment resulting from chronic or intermittent auditory tube obstruction. The greatest care, however, must be exercised in the selection of patients for irradiation and Bordley discusses the various factors which influence this therapy. In analyzing the results of irradiation of the nasopharynx in these patients suffering from a conductive type hearing loss, he is convinced that improvement of hearing impairment is more successful in children than in adults. Fifty per cent of carefully selected children in Bordley's experience have shown significant improvement in hearing following irradiation. The effectiveness of this type of therapy in simple aerotitis media, as reported by Haines, showed that 90 per cent of submarine trainees suffering from aerotitis resulting from their escape tank test were able to resume training after irradiation. Great reduction in the number and severity of upper respiratory infections has been noted following irradiation. Bordley states that if the technique and dosage prescribed by Burnam and Crowe is followed accurately, no bad results should be obtained from the use of the radium applicator in the nasopharynx.

In an editorial on the nasopharyngeal applicator, Jacox[35] states that clarification of some problems associated with this applicator may be needed because of the recent observations of Braestrup on the ionization measurements of the tissue dose in equivalent Roentgens. The dosage employed thus far has been more or less empirical. Studies have been published which indicate that it would be well to monitor every physician's technique to determine the number of treatments he may administer without exceeding the permissible value. Jacox states that if the standard applicator is used with the recommended precautions, there is little likelihood of any

serious damage. He points out that since it has been determined clinically that these exposures are safe and are necessary to obtain the desired results, radiologists should apply this knowledge of accurate ionization measurements in using the applicator in the future.

SURGERY.

Klopp and Delaney[36] point out that lesions of the hypopharynx, epiglottis, postcricoid region and base of the tongue are readily accessible through an anterior pharyngotomy. This procedure is simple, provides good exposure and does not interrupt any major vessel or nerve. A collar incision is made over the hyoid bone and the skin and platysma are undermined. The central portion of the hyoid bone is freed and a large segment removed. Incision of the pharyngeal mucosa then reveals a large area of the pharynx and hypopharynx and the base of the tongue for inspection and surgical procedure. The wound is carefully closed in layers without drainage. A perliminary low tracheotomy may be desirable. Postoperative healing is usually uncomplicated. Several illustrative cases are cited.

Farina[37] states that pharyngoneoplasties are indicated in patients in whom there is wide destruction of the pharynx. Pharyngectomies are generally performed on patients with cancer of the pharynx or advanced cancer of the larynx with invasion of the pharynx or perilaryngeal tissues requiring wide resection. Obviously, such extensive resection of tissue in the region of the neck would cause great discomfort because of constant discharge of salivary secretions which produce considerable local irritation of the skin. In addition to this, the patients experience much difficulty in swallowing and must be fed by a tube as long as the stoma is open.

The technique of the repair is well described and illustrated. Great stress is placed on the importance of nonsuperposition of suture layers to insure successful healing. Farina points out that the question of salivation and growth of hair in the interior of the pharynx no longer offers any serious obstacles

for such repairs. The use of atropine is discouraged. Satisfactory results have been obtained by Farina. The patient is able to obtain nourishment in the normal manner and his psychic state is greatly improved.

This article is all too short to cover such an important subject. An illustrative case report is included and it is hoped that more cases will be reported.

BENIGN TUMORS.

Schall[38] correctly calls attention to the fact that the nasopharynx is the most neglected area in laryngology. It is an area difficult to examine in many adults and in children it is best examined under general anesthesia. Lesions occurring in the nasopharynx present symptoms varying in size, location and extension of the tumor. Schall discusses all symptoms under five headings: nasal obstruction, tubal obstruction, hemorrhage, invasion of cranial nerves and metastasis. Attention is directed to benign nasopharyngeal fibromas which at times present serious problems in adolescents. These fibromas are frequently found in children five or six years of age and rarely after 20 years of age. The origin of nasopharyngeal fibromas is in the prevertebral fascia at the base of the sphenoid. Such tumors may completely fill the nasopharynx. In the treatment of the highly vascular tumors, Schall states that Herman and Figi use electrocoagulation, but he much prefers irradiation, either external or by implantation of radium needles into the tumor. Such articles as this help to make the laryngologist more conscious of the pathologic alterations which may develop in the nasopharynx.

Diehl[39] presents a case of nasopharyngeal fibroma in a woman aged 52 years. It was successfully removed surgically with little bleeding and was believed to be a fibrosed angioma or polyp.

Neurinomas are benign tumors arising from the neurilemmal sheath of peripheral nerves. They are firm and encapsulated and histologically show a palisading of spindle-shaped nuclei. They are rare in the pharynx, only about 15 cases

having been reported. Kemmom[40] presents a case in which a large neurinoma occurred in the lateral wall of the pharynx which was removed through a pharyngeal approach. The involved nerve often degenerates following removal of the tumor.

Hunter and Biggart[41] report the case of a large nasopharyngeal fibroma, in a boy 17 years old, which produced typical symptoms. Deep Roentgen therapy, tried first because of the difficulties of surgical treatment, produced no change in the tumor. Complete recovery followed surgical removal.

Figi and Davis[42] present an analysis of 51 cases of nasopharyngeal fibromas, also known as juvenile nasopharyngeal fibromas, seen at the Mayo Clinic from 1940 through 1949. This is the largest series reported in the literature.

These tumors always appear during adolescence, and although they may show regression when the patient reaches sexual maturity, Figi and Davis have never observed complete spontaneous disappearance. These tumors are benign histologically but may be highly destructive by local invasiveness. They occur predominantly in the male. All patients in the series reported were males. The average age of their patients when first seen was 15.1 years and most of them had had previous therapy. The clinical findings are consistent. Nasopharyngeal examination reveals a red, firm, well rounded mass occupying the nasopharynx. Ulceration is unusual. The tumor may also be seen by anterior rhinoscopy. Figi and Davis believe that the diagnosis can be made by the gross appearance of the mass and biopsy is contraindicated because of possible hemorrhage.

Treatment consists of electrocoagulation and implantation of radon seeds in most cases. One patient received electrocoagulation alone and seven, irradiation alone. Anesthesia is obtained by the intravenous administration of sodium pentothal and oxygen intratracheally. The tumor is approached through the nose and transorally through the nasopharynx and more recently transantrally through a Caldwell-Luc ap-

proach; in the latter instance the nasoantral wall is also removed. Secondary operations are often necessary and are done after an interval of six to eight weeks.

Of 42 cases with adequate follow-up, 72 per cent have been controlled. There were no immediate operative deaths in this series. One patient died of meningitis three months, and one of hemorrhage four months postoperatively.

This is an excellent and authoritative discussion of nasopharyngeal fibromas, and it would seem from the results obtained that the method of treatment described here is the procedure of choice.

Switzer and co-authors[43] describe a case of extramedullary plasmocytoma with several lesions in the pharynx, in addition to the skin and lymph node involvement, which failed to respond to all known types of therapy. These tumors are rare; their site of predilection is the upper respiratory tract. The clinically noncancerous stage may persist over 10 years, but it is ultimately fatal once the skeleton or lymph nodes become involved. Irradiation may cure isolated lesions.

Scevola[44] reports two cases of plasmocytoma of the rhinopharynx. The first case, a man aged 47 years with a fungating neoplasm on the lateral wall of the rhinopharynx, died two and one-half years after radiologic treatment with a large ulceration and glandular metastases. The other case, a man 38 years old, had a fungating neoplasm on the arch of the rhinopharynx, which was treated radiologically after incomplete surgical removal; he was in good health three years following treatment.

MALIGNANT TUMORS.

Malignancies of the nasopharynx rank third in incidence among neoplasms in Singapore. Severe pain in the frontal and parietal regions in the later stages is believed to be due to extradural extension of the growth. This pain often is not relieved by any drugs. In four cases described by Mekie and Ransome[45] bilateral leucotomy was done for relief of the pain.

One patient died several hours postoperatively. The remaining three obtained remarkable relief of pain and did not show mental deterioration.

In an excellent article, Villoria[46] thoroughly discusses the subject of nasopharyngeal malignancies. According to him, prior to 1900, nasopharyngeal malignancies were considered extremely rare; since then, however, many cases have been reported, and it is generally thought to be more common than reported cases indicate. Regaud, of France, was the first to recognize nasopharyngeal tumors as lymphoepitheliomas. About this same time Schmincke described a similar type of tumor which he said was extremely radiosensitive. It was determined that lymphoepitheliomas and lymphosarcomas constituted the most frequent types found in the nasopharynx. Villoria describes a case of lymphosarcoma treated radiologically. Many patients with nasopharyngeal malignancies are probably seen by capable otolaryngologists who fail to make a correct diagnosis. The signs and symptoms of nasopharyngeal tumors are well described. These tumors have a tendency to infiltrate the orbit, base of the skull and cranial vault. The only two posterior methods by which a representative piece of tissue may be obtained for microscopic examination are either through the nose or by elevating the soft palate. The generally accepted therapy for nasopharyngeal cancer is radiation, as most of these tumors are radiosensitive. Villoria reports a case of plasmocytoma of the nasopharynx and practically reviews the literature on this tumor.

Owen[47] discusses at length the problem of hypopharyngeal carcinoma. The importance of early diagnosis is emphasized, including the significance of a collection of mucus in the pyriform fossa on mirror examination and esophagoscopy in cases of dysphagia. He believes that careful follow-up and repeated esophagoscopies are indicated in women with dysphagia and anemia, which he calls "Patterson-Brown Kelly syndrome." Of 34 such patients followed five years or more, postcricoid carcinoma developed in five. Owen prefers surgical treatment to Roentgen therapy. He differentiates the soft ulcerative type of postcricoid carcinoma which metastasizes early from

the scirrhous type. He advises pharyngolaryngectomy with primary closure of the pharyngostomy with a skin graft for the ulcerative type, but believes that a lateral pharyngotomy or transthyroid pharyngotomy may be sufficient in the scirrhous type.

This article presents a good discussion of the problem of postcricoid carcinoma but adds nothing new to the treatment of this most distressing condition.

According to Eberhard and Leaming,[48] the ideal goal of radiotherapy for nasopharyngeal malignancies is a minimum of 4,000 to 4,500 r. to all parts of the tumor in four weeks. This is best accomplished by the use of two lateral fields and two antral fields, and in addition cervical fields when cervical metastases are present. All types of tumor should receive the same dosage when possible. Lymphosarcomas, which comprise 40 per cent, and lymphoepitheliomas, which comprise 10 per cent of malignant tumors of the nasopharynx, are more radiosensitive. In patients without bony involvement, about 40 per cent may survive five years; with bony involvement, only about 10 per cent will survive this period. An overall salvage rate of about 20 per cent should be expected from all patients treated by irradiation.

Reaction to treatment can be severe and medical management is of utmost importance. Occasionally because of a severe cutaneous reaction it is necessary to use radium locally rather than Roentgen-ray therapy.

This article contains exact descriptions of technique used which will be of interest to radiotherapists.

Quite a few cases of pituitary chromophobe adenomas have been reported in the nasopharynx, but Kay and co-authors[49] state that three cases described in this article are the first cases reported involving the nasal cavity. The diagnosis of all three cases was made by radiologic evidence of enlargement of the sella. These tumors do not metastasize but may be locally malignant and invasive. In each case nasal obstruction and rhinorrhea occurred. Rhinoscopic examination revealed

reddish polyps in the postethmoid region in two cases and in the middle meatus of the third. The growths were partially removed intranasally in each case; the pathologic diagnosis was pituitary chrombophobe adenoma in two cases and it was questionable in the third. All three patients received Roentgen therapy and are living 14 years, five years, and 18 months after treatment.

Cunningham and Hu[50] briefly review the literature on nasopharyngeal cancer in the Chinese to direct attention to the prevalence of this disease in the Chinese between the ages of 30 and 40 years. An analysis of 37 cases which were encountered between 1944 and 1947 in the teaching hospitals of the West Chinese Union University showed that males were affected three times as often as females. In their series the most obvious sign was painless enlargement of the cervical nodes of the spinal accessory chain in one or both posterior triangles of the neck. Other clinical manifestations which indicate a possible malignant lesion in the nasopharynx are nasal bleeding, tinnitus and deafness, numbness over the maxillary region, paralysis of the VIth or IIIrd nerves, unilateral headache and trismus. Cunningham and Hu urge a nasopharyngeal examination for every patient with symptoms relating to the eye, ear, nose and neck.

Kramer[51] analyzed 54 cases of malignancy of the nasopharynx treated at Middlesex Hospital from 1935-1949. These tumors account for 0.7 per cent of the deaths from cancer in England.

Symptoms of these patients may be divided into three groups: symptoms due to presence of the tumor locally, those due to intracranial extension through the foramina of the skull, and those due to metastatic lesions, principally cervical nodes. Seventy-six per cent of the 54 patients in his series had cervical metastasis and 25 per cent had involvement of the cranial nerve when first seen.

Kramer differentiates squamous cell carcinoma, anaplastic carcinoma, transitional cell carcinoma, and lymphoepithelioma as well as the nonepithelial malignancies. It is of interest to

note that over 50 per cent of these cases with cranial nerve involvement were transitional cell carcinomas; however, there is still much discussion among pathologists about the differentiation of the various types of malignancies occurring in this region.

The five-year survival rate in Kramer's series was 25 per cent. All patients were treated by Roentgen-ray therapy. Transitional cell carcinoma has the worst prognosis. Most of the patients surviving two years will be alive without recurrence at the end of five years.

In a clinicopathological conference[52] the clinical symptoms indicated the presence of a tumor of the nasopharynx invading the maxilla and the orbit. Biopsy from this tumor, which could be seen protruding from the nasopharynx, was reported as acute and chronic inflammation. A medical man expressed surprise that he was asked to conduct the differential diagnosis instead of an otolaryngologist. A small ulcerative lesion on the scrotum proved to be a carcinoma and the Roentgenograms showed a questionable shadow in the mediastinum. The white blood count was 56,000 per 100 cc. The disease was rapidly fatal, and autopsy revealed squamous cell carcinoma of the nasopharynx, maxilla and orbit. A carcinomatous mediastinal tumor was found to have perforated into the wall of the superior vena cava, formed a thrombus and extended down into the auricle. The tumor invaded the superior segment of the left lung and the lumen of the bronchus showed involvement. The question naturally arose as to which was the primary tumor. The pathologist expressed the opinion that this was a primary tumor of bronchogenic origin with metastasis to the nasopharynx, sinuses and cervical region.

Such cases as this are of extreme interest to the otolaryngologist because they vividly demonstrate that at no time can we afford to confine our study only to the domains of otolaryngology. The symptoms described are typical of tumors of the nasopharynx and an alert otolaryngologist should have discovered the lesion long before it reached such alarming proportions. This case further demonstrates that a negative

biopsy report, in patients with clinical evidence of malignancy, such as cervical metastasis, is of no value and repeated biopsies should be done until representative tissue is obtained. Discussion of such cases is not only instructive but makes the otolaryngologist more conscious of disease of the nasopharynx.

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CAPILLARY AREAS OF THE COCHLEA IN THE GUINEA PIG.

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Interest in the vascular system of the inner ear was stimulated about the middle of the nineteenth century by the publication of the first histological descriptions of the end-organs within the membranous labyrinth. Investigators were impressed with the importance of understanding the circulation in such a highly differentiated structure and numerous attempts to solve the intricacies of the vascular configuration followed.

Most of the early investigators (Huschke,[1] Retzius,[2] Voltolini,[3] Boettcher[4]) noted the presence of blood vessels related to the inner ear structures which they studied and described their course and position, but Schwalbe[5] (1887) was the first to coordinate the circulation of the entire cochlea. The coiled arterioles of the modiolus particularly interested him, and he gave little attention to the capillaries. He was followed, in 1892, by Eichler[6] who introduced the technique of the intravascular injection of a dye and preparation of a cleared celloidin cast of the ear. While his studies on the human added little to Schwalbe's earlier work on the guinea pig, his methods were used in most subsequent investigations of the labyrinthine circulation.

Siebenmann's[7] illustrated monograph, published in 1894, became the classical study. He presented a clear picture of the general features of the circulatory pattern in man and

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emphasized the complete separation of the blood supply of the membranous labyrinth from that of its bony capsule. The capillaries in the spiral lamina and spiral ligament were shown as loosely organized networks.

Shambaugh,[8] in 1903, was the first to recognize an orderly arrangement of the vessels in the outer wall of the cochlea in the pig. He divided them into three distinct capillary areas. Further comparative studies by Asai,[9,10] in 1908, on the dog and the rat revealed nothing new except a similarity in the vascular pattern of these animals to that of man.

In 1923, Nabeya[11] published an extensive study of the labyrinthine circulation of the mammalian ear. He carefully outlined the more general vascular characteristics but added little to our knowledge of the capillary regions.

In recent years, only a few studies on specific capillary areas have been made. Belemer[12] (1936) reported that the vessels entering and leaving the stria vascularis were arterioles. In 1949, Agazzi,[13] investigating the vascular hydrodynamics of the spiral ligament in the guinea pig, divided the capillaries in this region into two separate systems.

Each one of these investigators has contributed something toward a better understanding of the circulatory pattern, but the study remains incomplete. Interest has been primarily centered about the larger features. The capillaries, in most cases, have been indistinctly shown and inadequately described. Little attempt has been made to locate them precisely in relation to various parts of the inner ear.

The necessity for further studies on certain vascular areas in the ear has been emphasized in the past few years. Recent reports have shown that there is still no clear concept of the capillary arrangement in the spiral ligament. The question of the origin and absorption of endolymph and perilymph is one that would profit from a more detailed analysis of the distribution of blood vessels. Ménière's disease, tinnitus, and some hearing disorders have been attributed to vasomotor disturbances in the cochlear and vestibular labyrinth. It is important

that the details of capillary ramifications and relations be as clearly outlined as have other features of the vascular pattern. It is possible that a study of the capillary areas of the cochlea may add something to our knowledge of normal or pathological circulatory conditions.

MATERIALS AND METHODS.

Seventy-five guinea pigs weighing from 250 to 450 gm. were used. They were healthy animals of dark color with good pinna reflexes.

The vascular pattern was studied by means of the intravascular precipitation of Prussian blue or lead chromate. It was found that this method resulted in more complete capillary injections than perfusion of a dye or particulate material.

The animals were given 0.6 ml. veterinary Nembutal (Pentobarbitol Sodium, Abbott), the chest cavity was opened and a cannula inserted in the aorta. The blood was first washed out with 0.85 per cent sodium chloride solution or Locke's solution at approximately normal blood pressure.

An intravascular precipitation of Prussian blue was induced in 17 guinea pigs. This was accomplished by perfusing first with 75 to 100 ml. of 2 per cent of equal parts aqueous potassium ferrocyanide and iron ammonium citrate. A small amount (25 to 50 ml.) of distilled water was then run in to avoid precipitation in the tubing, followed by 50 to 75 ml. of 3 per cent hydrochloric acid in 10 per cent formalin solution. The temporal bones were removed from the animal, the cochleae were exposed and the specimens allowed to remain in the acidified formalin overnight. Some of these ears were completely decalcified, embedded in paraffin and sectioned. Most of the ears, after washing well, were dissected under the dissection microscope at magnifications of 18× to 54×. Pieces of the inner ear were removed as desired. They were counterstained lightly with 0.2 per cent aqueous Safranin O solution, dehydrated, cleared and mounted in Damar for microscopic examination.

The method of Williams[14] was followed for an intravascular precipitation of lead chromate crystals in eight guinea pigs. This method was found to be most suitable for visualization of the vascular pattern and topography of the cochlea as a whole under the dissection microscope at magnifications of 18× to 100×.

The cytology of the vessel walls was studied by the examination of dissected pieces of tissue stained with 0.2 per cent aqueous Safranin O, alone or combined with Light Green, and sectioned material stained by hematoxylin and eosin.

Bodian's protargol method (Silver albumose, Winthrop) was used on dissected and sectioned material to demonstrate the presence of nerve fibres. Eleven animals were perfused with 10 per cent formalin solution and the ears fixed for two days. Half of the ears were decalcified with 2.6 per cent nitric acid in 12 per cent formalin solution; the others were stained in the undecalcified state. The cochleae were opened, and the cochlear duct and blood vessels in the modiolus were exposed before silver impregnation. After impregnation the blood vessels were dissected out from the modiolus, pieces of spiral ligament and spiral lamina removed, and the staining procedure completed.

Sections of paraffin embedded ears were stained by Weil's iron hematoxylin method to differentiate between the myelinated fibres of the cochlear nerve and the unmyelinated fibres of the blood vessels.

FINDINGS.

The general features of the vascular supply has been previously described;[7,11] therefore, they will be outlined only briefly before going on to the detailed description of the capillary areas.

The labyrinthine artery enters the internal auditory meatus, gives off the A. vestibuli anterior, then continues to the base of the modiolus as the A. cochleae communis. Here it divides into the A. vestibuli posterior and the A. cochleae propria.

The A. cochleae propria enters the modiolus with the cochlear nerve. It gives off numerous branches in its spiral ascent to the top coil and ends somewhere in the fourth turn by dividing into two branches, one of which supplies the spiral ligament of the apex.

In its course upward, the A. cochleae propria is embedded in the loose connective tissue between the rope-like core of nerve fibres and the bony wall. In this position it gives off large primary branches which in turn ramify into secondary branches, or smaller branches equivalent to the latter. The primary branches may coil or twist a few times in the loose connective tissue before dividing further. The first primary branch is notable in that it is a large branch, often supplying a part of the basal turn and the vestibular portion of the cochlear duct, anastomosing with branches from the posterior vestibular artery.

The secondary radiating branches coil tortuously in bone before arching out over the scala vestibuli. They strike outward to enter a wedge-shaped plate of bone located superior and medial to the scala vestibuli, and inferior to the posterior spiral vein. In the bone, they assume the general pattern of a spring coil directed toward the bony partition; however, the coils are not completely enclosed in bony channels. At the root of the bony partition the arterioles straighten out, arching over the scala vestibuli enclosed in a bony canal for a variable distance to enter the spiral ligament at its uppermost attachment.

The distance for which the radiating arteriole traverses the bone usually decreases as the apex of the cochlea is approached. In the first turn the radiating vessel is encased in bone across the top of the scala, descends along the lateral edge and at times does not leave the bone until below the upper limits of the spiral ligament. In the second and third turns, the vessel leaves the bone after it passes the lateral limits of the bony partition. In the fourth turn and the apex, it may not be enclosed in bone for the greater part of its course. Wherever the vessels leave the bone before entering

the spiral ligament, they are separated from the perilymphatic space by only the thin layer of mesothelial cells which lines the scalae.

I — Capillaries of the Spiral Ligament.

Although it may divide into two branches in the bony partition, the arteriole does not ramify into terminal branches until it enters the spiral ligament. One or two small branches turn and course in a spiral direction; the remainder descend in the spiral ligament. These capillaries of the spiral ligament may be divided into four groups on a basis of their location and course. Group 1 consists of the branches that wind in a spiral direction in the upper spiral ligament. Group 2 is the capillary network in the stria vascularis. Group 3 is the vessel in the spiral prominence. Group 4 includes the remaining capillaries which descend in the depths of the spiral ligament (see Figs. 1 and 2).

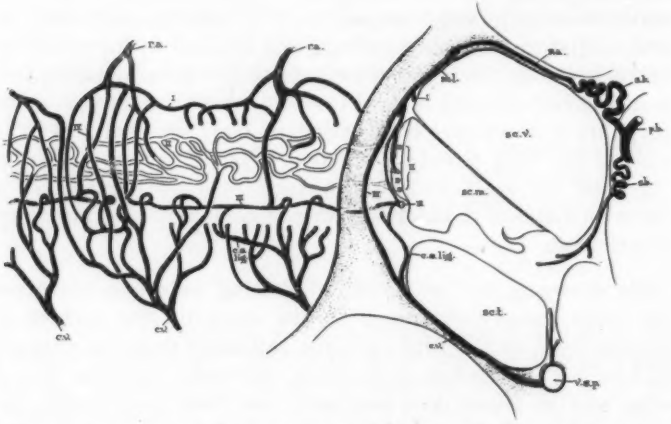

Fig. 1. Photograph of a schematic drawing showing typical capillary ramifications in the spiral ligament of the guinea pig. (c.s.lig.) crest of the spiral ligament; (c.v.) collecting venule; (m.l.) mesothelial layer; (p.b.) primary branch; (r.a.) radiating arteriole; (s.b.) secondary branch; (sc.m.) scala media; (sc.t.) scala tympani; (sc.v.) scala vestibuli; (V.sp.p.) V. spiralis posterior; (I) capillary of the upper spiral ligament; (II) network of the stria vascularis; (III) vessel of the spiral prominence; (IV) capillary descending in the depths of the spiral ligament.

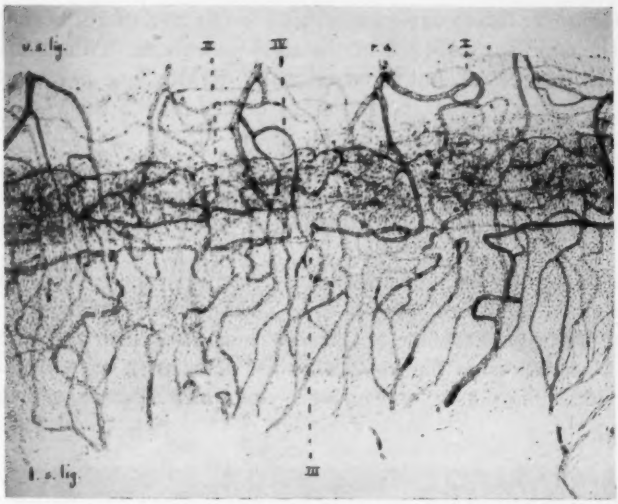

Fig. 2. Photomicrograph of dissected specimen of spiral ligament from guinea pig No. 79. Intravascular precipitation of Prussian blue; Safranin O stain; magnification 100×. (l.s.lig.) lower spiral ligament; (r.a.) radiating arteriole; (u.s.lig.) upper spiral ligament; (I) capillary of the upper spiral ligament; (II) network of the stria vascularis; (III) vessel of the spiral prominence; (IV) capillary descending in the depths of the spiral ligament.

Group 1: One or two of these small branches are given off directly from each radiating arteriole or from one of its terminal ramifications. They pursue a winding course in a spiral direction, usually above the attachment of Reissner's membrane, although in their undulations they sometimes dip below it. Lying just below the surface of the mesothelial lining of the scala vestibuli, they may cross one, two or three arterioles and their branches and anastomose with other tributaries. They leave the upper spiral ligament in one of two ways: they usually turn downward and descend with the other capillaries in the thicker part of the spiral ligament, joining the venules below; or they turn upward, crossing the radiating arterioles diagonally in the bone to pierce the bony partition and end in the collecting vein of the turn above.

Group 2: Each arteriole usually sends one of its branches to the capillary network of the stria vascularis. This is often a terminal vessel, but it occasionally divides, one part entering the stria vascularis, the other descending to the vessel of the spiral prominence or the lower spiral ligament. The branch descends in the spiral ligament behind the stria vascularis, usually entering the strial epithelium in its upper half to empty into the vascular plexus.

The capillaries of the stria vascularis pursue a serpentine course in the spiral direction in between the epithelial cells (see Fig. 3). They are extensively connected with each other so that they give the appearance of a network or wide one-layered honeycomb superimposed upon and disposed at right

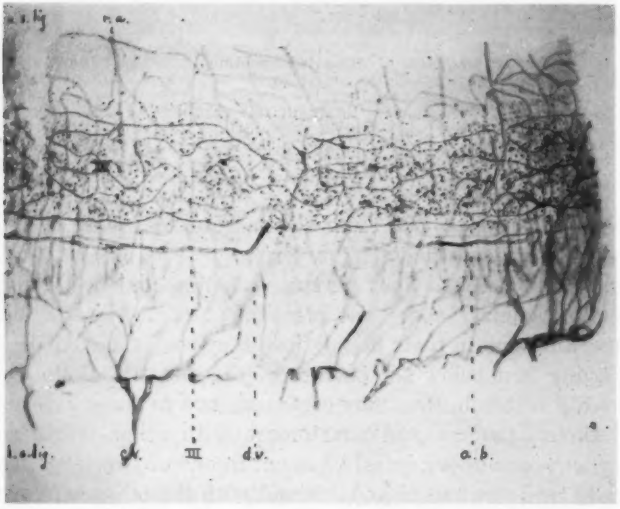

Fig. 3. Photomicrograph of dissected specimen of spiral ligament from guinea pig No. 21 showing network of the stria vascularis. Intravascular precipitation of Prussian blue; Safranin O stain; magnification 100×. (a.b) arteriolar branch entering the stria vascularis; (c.v.) collecting venule; (d.v.) venule draining the strial network; (l.s.lig.) lower spiral ligament; (r.a.) radiating arteriole; (u.s.lig.) upper spiral ligament; (II) network of the stria vascularis; (III) vessel of the spiral prominence.

angles to the deeper vessels of the spiral ligament. The superior and inferior boundaries are comparatively straight and parallel.

The strial rete is drained by a venule of large caliber. It is formed by the junction of three or four of the strial capillaries and turns backward to leave the stria vascularis in its lower half. It descends peripherally to most of the other descending vessels and may receive a branch or two from them before entering into the collecting venous system at the lower edge of the spiral ligament.

In all of the animals observed, no other connection was noted between the strial capillaries and other capillaries in the spiral ligament except through the arterial branch entering and the venule leaving the network.

Group 3: Another of the main branches usually descends behind the stria vascularis and curves inward to enter the spiral prominence, where it empties into a spirally coursing vessel. Each radiating arteriole usually supplies one branch to the vessel of the spiral prominence, although there may be more than one, or none. The small capillaries of the first group seldom end here.

The vessel in the spiral prominence is found near the epithelial layer, sometimes with only a single layer of connective tissue cells interposed. It courses parallel to the network of the stria vascularis, just below its inferior border, but in no way connected with it. It is not continuous, rarely continuing for more than 1 or 2 mm. without a break. At times it is double, a duplicate vessel running for a short distance up under the edge of the stria vascularis.

This capillary is drained by many vessels, always two or three times the number of those entering it. They must bend upward and backward to leave the spiral prominence because of interference below from the outer spiral sulcus. They then descend toward the collecting venules of the lower spiral ligament.

Group 4: The remaining terminal branches descend in the spiral ligament, some curving out toward the periosteum. At times they divide in the depths of the tissue. This is found most often in the basal turn, where the tissue is thicker. Ordinarily there are no anastomoses between the capillaries descending in the spiral ligament until they reach the area below the basilar membrane.

Some of the capillaries descending close to the stria vascularis bend toward the scala tympani at a point from 30 to 50 microns below the level of the basilar membrane. They turn and continue in a spiral direction for 50 to 400 microns, sometimes receiving other of the descending vessels. In the basal turn this part of the scala tympani is covered by a thin plate of bone. Elsewhere, the vessels lie close to the mesothelial lining of the scala, with only a few descending basilar membrane fibres intervening. When the capillaries again turn downward, they continues to the collecting venule, lying below the mesothelial layer.

The other descending capillaries, following the curvature of the bone, turn slightly inward to meet the vessels just described, those from the strial rete and the spiral prominence. They are all united in the venules of the lower spiral ligament. At the inferior boundary of the scala tympani, they have been brought together into two or three trunks. These then cross the floor of the scala tympani, slanting in a basal direction, uniting into one large vein. Corresponding to the situation of the radiating arteriole, the venule does not enter the bone of the floor of the scala in the upper turns until it has almost reached the posterior spiral vein.

The V. spiralis posterior receives all the venous blood from the cochlea. It begins in the apex by receiving venules from the spiral lamina and spiral ligament, and makes a spiral descent at the inferior medial border of the scala tympani. As it descends it receives veins radiating inward under the scala tympani from the spiral ligament, those descending from the spiral lamina and others from the spiral ganglion and modiolus. At the proximal end of the basal coil, it turns and enters the V. canaliculi cochleae at the mouth of the cochlear

aqueduct. The V. fenestrae cochleae and the V. vestibuli posterior, both of which may collect a few venules from the vestibular portion of the cochlear duct, receive drainage from the remaining part of the membranous labyrinth. They empty into the V. canaliculi cochlea which terminates in the jugular vein.

II — Capillaries of the Spiral Lamina (see Fig. 4).

The vessels supplying the spiral lamina originate either from the primary branches of the A. cochleae propria or the

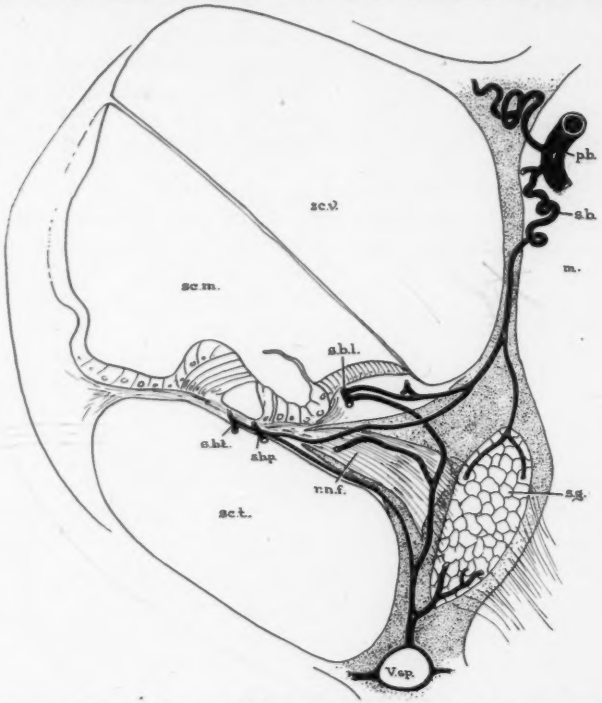

Fig. 4. Photograph of a schematic drawing showing typical capillary ramifications in the spiral lamina of the guinea pig. (m) modiolus; (p.b) primary branch; (r.n.f.) radial nerve fibres; (s.b.) secondary branch; (s.b.l.) spiral border of the limbus; (s.b.p.) spiral border below the inner pillar; (s.b.t.) spiral border below the tunnel; (s.g.) spiral ganglion; (sc.m.) scala media; (sc.t.) scala tympani; (sc.v.) scala vestibuli; (V.sp.p.) V. spiralis posterior.

proximal end of the secondary coiled branches. They may coil a few time in the modiolus, then descend toward the spiral lamina. They turn outward in the upper plate of the bony spiral lamina to slightly beyond the medial limits of the limbus. If they have not previously branched, they divide here in various ways. One tributary may ascend into the limbus, while another continues outward in the bone. At times the vessel divides into two divergent branches coursing spirally at the root of the limbus, sending capillaries into the limbus and finally entering there itself.

The capillary bed of the spiral lamina is divided into two regions for descriptive purposes: an upper plexus in the limbus, a lower plexus below the basilar membrane. Each plexus may have separate arterial supply and venous drainage, although some capillaries are continuous between the two.

Vessels of the Limbus: The vessels enter the limbus by passing into the connective tissue base near its medial attachment. They ascend diagonally toward the lateral edge of the epithelial covering. The capillaries do not enter the epithelial layer, nor the fibrous area bordering the internal spiral sulcus but remain in the connective tissue base of the limbus. At the superior lateral edge of the connective tissue. inferior to the epithelial layer, the vessels turn and form an irregular edging of loops (see Fig. 5).

From the upper edge of the limbus, the vessels descend in the same manner through the connective tissue to re-enter the upper plate of the spiral lamina. In the bone, the capillaries either continue alone toward the vein or join the descending vessels from the lower plexus.

Vessels Below the Basilar Membrane: The arteriolar branch to the plexus situated below the basilar membrane ordinarily approaches in one of two ways: either by descending in the bone of the modiolus and continuing out in the bony spiral lamina after giving off a tributary to the limbus, or by descending in the central canal of the modiolus: The first manner of approach has been described. The second manner of approach is found in the first turn, part of the second and

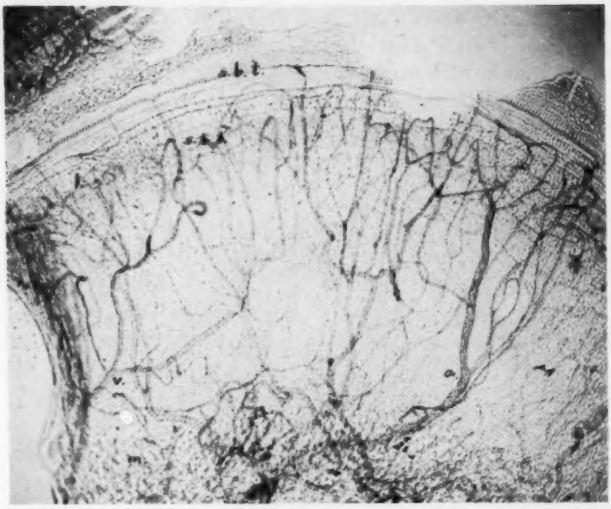

Fig. 5. Photomicrograph of dissected specimen of spiral lamina from guinea pig No. 66 showing the capillaries of the limbus. Intravascular precipitation of Prussian blue; Safranin O stain; magnification 85×. (a) arteriole; (l) limbus; (m) modiolus; (s.b.l.) spiral border of the limbus; (s.b.t.) spiral border below the tunnel; (v) venule.

infrequently elsewhere. The arteriole twists as it descends in the modiolus, then pierces the superior medial corner of the bony wall of Rosenthal's canal. Here, or before, branches may be given off to the limbus and spiral ganglion.

Whatever the manner of their approach to the upper plate of the spiral lamina, they extend outward either enclosed in the bone or placed directly on top of the radial fibres of the cochlear nerve. Here the vessels are in intimate contact with the myelinated fibres. In spreading out they sometimes dip under a few fibres. They descend to the lower plate of the spiral lamina through the nerve, usually traversing the interspaces, but at times penetrating the bundles.

Having reached a position inferior to the nerve fibres, the vessels continue out beyond the bone and below the fibres of the basilar membrane. Below the inner pillar of the organ of

Corti, the capillaries turn in a spiral direction and form an interrupted border (see Fig. 6). Although indentations are present, this border appears much straighter than that of the

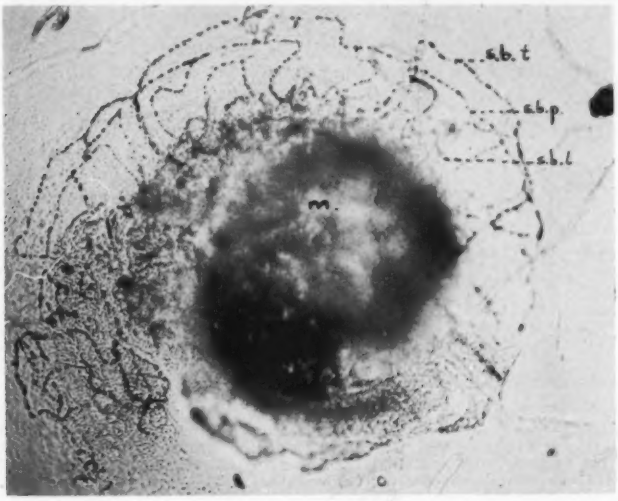

Fig. 6. Photomicrograph of dissected specimen of spiral lamina of the fourth turn from guinea pig No. 67 showing the capillaries below the basilar membrane. Intravascular precipitation of lead chromate; magnification 100×. (m) modiolus; (s.b.l.) spiral border of the limbus; (s.b.p.) spiral border below the inner pillar : (s.b.t.) spiral border below the tunnel.

limbus. This border has many radial connections of a diverse nature. Approximately one-half of its tributaries descend directly toward the posterior spiral vein. Of the other half, some are arterial and some are so joined with the limbus vessels that it can only be said that they are part of the capillary bed.

The second spiral border located inferior to the basilar membrane fibres is found below the tunnel of the organ of Corti (see Fig. 6). This vessel has fewer tributaries than the vessel below the inner pillar, the ratio being about 1:3 or 1:4. Many of its branches are capillary bridges between the

two spiral borders; however, it does have direct arterial and venous ramifications from the modiolus, particularly in the first turn.

Both of these spiral borders situated below the basilar membrane are separated from the perilymphatic space by the mesothelial lining and a thin layer of basilar membrane cells.

The blood is returned to the posterior spiral vein by one of two pathways. The vessels extend back up through the nerve fibres, descending at the lateral border of the spiral ganglion, or they descend following the curvature of the scala tympani. The vessels descending along the scala tympani may be enclosed in bony canals or be covered only by the mesothelium lining the fluid space. In some cases the departing vessel connects the two infrabasilar membrane borders, then follows the lower plate of the spiral lamina, but outside the bone. It continues for about half the extent of the bony lamina, then pierces the bone and completes its descent to the posterior spiral vein within a bony channel. In other cases the venule leaving the spiral borders enters the bone, descending therein to the vein.

The other pattern of venous drainage shows the capillaries re-entering the spiral lamina with the nerve fibres, slanting up through the fibre bundles and continuing medially to the lateral edge of the spiral ganglion. Here they may join the postcapillary venules from the limbus before descending in bone or through the nerve fibres.

All of these are collected together in venules which also receive branches from the spiral ganglion, to end in the posterior spiral vein. At the apex, the spiral vessels of limbus and basilar membrane are brought together in the first venules of the posterior spiral vein. In the vestibule, they likewise are joined in a branch of the posterior vestibular vein.

III — Capillaries of the Spiral Ganglion and the Modiolus.

The arteriolar supply to the spiral ganglion comes from either a primary branch of the A. cochleae propria or the

arteriole descending to the spiral lamina. The vessels enter Rosenthal's canal from above and intertwine freely among the ganglion cells. There is little anastomosis with the capillaries leaving the basilar membrane and limbus. At times the vessels are collected together in the same postcapillary venules. Otherwise the venules from the spiral ganglion enter the posterior spiral vein by a separate collecting venule.

The arterial supply to the spiral ganglion appears to be entirely separate from that to the fibres in the central canal of the modiolus. In the proximal part of the first turn, the fibres leaving the spiral ganglion are enclosed in bone for a short space from spiral lamina to central canal. The vessels supplying these fibres sometimes make connections with the spiral ganglion vessels and even those leaving the basilar membrane; however, in many cross-sections observed, no vessels were ever noted crossing the bony plates between the ganglion cells and modiolus, or associated with the fibres leaving the spiral ganglion, other than the exception above noted.

The spiral nerve fibres in the central canal of the modiolus are well supplied by vessels coming directly from the artery or from the primary branches. They are highly anastomotic and uncoiled. The collecting venules enter the posterior spiral vein from the modiolus.

IV — Cytology of the Vessel Walls.

The thin vessel wall of the A. cochleae propria in the central canal of the modiolus contains two or three layers of longitudinal and transverse smooth muscle cells around the endothelial lining, plus a narrow adventitia. Its tributaries, decreasing in size, have a relatively less dense muscularis. As the vessels coil outward in the bone, the muscle cells are found to be more and more sparsely spread.

The arteriole radiating out over the scala vestibuli consists of an endothelial lining, with an occasional tangental or longitudinal—more rarely transverse—smooth muscle cell around the endothelial layer and a thin adventitia of two or three layers of connective tissue cells arranged parallel to the ves-

sel. A small accumulation of transverse smooth muscle cells can sometimes be observed in the fork or around the arteriole as it divides in the upper spiral ligament. The perivascular cells are divided between branches so that each main branch is usually enclosed in one layer of connective tissue cells, longitudinally disposed. When the main branches again divide, the connective tissue coating is lost.

The capillaries are composed of endothelial cells and an infrequent smooth muscle cell. This is typical of all of the capillaries of the spiral ligament except those in the stria vascularis. As the collecting venules leave the spiral ligament, a connective tissue adventitia is once more added.

The capillaries of the strial network appear to be composed only of endothelial cells. In some preparations, cells were seen which seemed to be applied to the outside of the capillary wall, but whether they were smooth muscle cells or closely applied epithelial cells could not be determined with certainty.

The vein draining the strial rete, as before noted, is of large caliber. It was observed at times to have one, two or three smooth muscle cells arranged transversely or tangentally across its mouth. The venule obtains a connective tissue perivascular layer as it leaves the stria vascularis. This is not characteristic of other vessels at this level.

The vessels supplying the spiral lamina are similar in appearance to those of the spiral ligament. The arterioles approaching the spiral lamina gradually lose most of their smooth muscle coating. As they enter the limbus and Rosenthal's canal, they are reduced to capillary size. The capillaries extending throughout the spiral lamina are endothelial walled tubes with occasional smooth muscle cells applied about them, particularly at bifurcation points. The vessel below the tunnel appears to contain fewer contractile elements than the other capillaries; however, the oval nuclei of the cells of the basilar membrane are closely applied to this vessel, at times giving impression of being directed around the vessel wall.

The capillaries in the spiral ganglion and those supplying the nerve fibres in the modiolus are likewise endothelial walled tubes, with occasional smooth muscle cells.

V — Innervation of the Blood Vessels.

Many nerve fibres accompany the labyrinthine artery entering the internal auditory meatus. Some are found in bundles contained in a connective tissue syncytium. Numerous naked nerve fibres can also be seen, either in the vessel wall or closely applied to it as it enters the meatus. The fibres are unmyelinated and seem to have no connection with the cochlear nerve.

The fibre bundles become smaller as they follow the artery up into the modiolus, branches being sent out from the bundles to the artery and its primary and secondary tributaries. The nerve fibres were found on the coiled secondary branches of the cochlear artery, but no fibres were demonstrable with the arterioles radiating out in the bony partition, or descending in the bone of the modiolus to the spiral lamina; neither were any nerve filaments observed with the capillaries.

No definite specialized endings could be observed. As the fibres become closely applied to the vessel wall, they lose any sheath in which they were previously enveloped and disappear as naked fibres.

DISCUSSION.

The capillaries of the spiral lamina have received consideration previously by all investigators, and little can be added concerning the general pattern. Attention has been directed primarily to the spiral vessels which have been described in various places in the spiral lamina. Shambaugh[8] objected to the term "spiral vessel." He emphasized that these were borders of capillary areas formed by capillary loops anastomosing with each other. Nabeya[11] recognized a similar condition in the human, designating only the vessel below the tunnel as a spiral vessel. "Spiral border" seems to be a preferable term,

as "spiral vessel" infers a vessel continuous from base to apex. The spiral borders found in the limbus and below the basilar membrane are discontinuous and often present a looped appearance.

The relation of the two lower spiral borders to the basilar membrane should be clarified. Retzius[2] stated that the spiral vessel below the tunnel was located in a layer of basilar membrane cells below the basilar membrane fibres. Boettcher[4] described the vessel under the tunnel as being below the basilar membrane. Siebenmann[7] located it in the basilar membrane. Asai[9] agreed with Siebenmann. Nabeya[11] found two spiral vessels on the basilar membrane. Observations made on the present series also revealed two spiral borders, one below the inner pillar cell, and one under the tunnel. They were found to be beneath the basilar membrane fibres with one exception. The vessel below the tunnel in the basal turn of some guinea pigs seemed to have a thin layer of basilar membrane fibres coursing beneath it, so that the vessel was between the fibres. This situation was observed only in the first turn.

A description of all the capillary ramifications of the spiral ligament has not previously been given. Shambaugh,[8] in 1903, first showed a definite arrangement for the capillaries of this region in the pig. He recognized a capillary area over the scala vestibuli which he believed to be intraosseous, another in the stria vascularis, and a third in the deeper part of the spiral ligament. He described the strial capillaries as a continuous system of loops, separate from the other vessels in this area. Nabeya[11] believed the capillaries of the stria vascularis to be superimposed upon those of the spiral ligament. Agazzi[13] divided the vessels of the spiral ligament into two systems: one, the capillary rete of the stria vascularis, the other consisting of the short, direct vessels in the depths of the spiral ligament.

The capillaries of the spiral ligament can actually be classified into four groups: one in the upper spiral ligament, one in the stria vascularis, one in the spiral prominence and a

fourth in the depths of the spiral ligament. A typical secondary radiating arteriole divides in the upper spiral ligament and sends one or two branches to the first group, one to the stria vascularis, one to the spiral prominence and several to the last group.

The small capillaries in the upper spiral ligament wind in a spiral direction and are not intraosseous as Shambaugh[8] found them to be in the pig. In the guinea pig they are situated just below the mesothelium lining the scala vestibuli. Some of them do traverse the bone as they ascend to the vein of the turn above.

The capillary network in the stria vascularis usually has one arteriolar branch entering it and a venule leaving it at approximately equivalent intervals. It has no other connection with the capillaries of the spiral ligament. This was indicated by Shambaugh[8] and Agazzi.[13] The system is composed of a closely meshed band of capillaries continuous from vestibule to apex.

The vessel of the spiral prominence has been given little attention. Shambaugh[8] included it in his illustrations as a part of the strial rete. Nabeya[11] noted the presence of a vessel in the spiral prominence but did not describe it, while Agazzi[13] did not include it in his model of the vessels in the spiral ligament. It was found in these studies that the vessel in the spiral prominence was a constant feature. While it is not continuous from base to apex, it usually can be found coursing straight through the prominence for 1 to 2 mm. without a break. Although it courses just below and parallel to the inferior border of the strial rete, it has no connection with the network of the stria vascularis.

The vessels descending in the spiral ligament were designated by Agazzi[13] as short, direct arteriovenular arcades. This applies to all except those that turn in the spiral direction for a short distance in the crest of the spiral ligament. Nabeya[11] described a spiral vessel in this area in the guinea pig. Asai[9] also noted a spiral vessel in the crest of the spiral ligament of

the dog. Siebenmann[7] believed it to be an inconstant vessel with a spiral course. Shambaugh's[8] observations are in close agreement with those of this study. No spiral vessel was observed in this area for the guinea pig. Some of the capillaries descending in the depths of the spiral ligament curve toward the scala tympani. Reaching the surface of the crest of the spiral ligament, they turn to course in a spiral direction for 100 to 400 microns, sometimes receiving branches from other capillaries. Eventually, they descend to the venules.

Few observations have been made as to the structure of the vessel walls. One of the early investigators, Schwalbe,[5] described the smooth muscle coating of the coiled secondary branches in the modiolus. Little has been said about the vessels elsewhere. Belemer[12] reported that vessels entered and left the stria vascularis as arterioles. Agazzi[13] called the vessels in the depths of the spiral ligament arteriovenous arcades but specified that they were endothelial walled tubes unprovided with smooth muscle cells.

The capillaries in the spiral lamina and the spiral ligament with the exception of those in the stria vascularis, all revealed scattered smooth muscle cells. Only the capillaries of the strial network showed no definite contractile elements. These appear to be composed only of endothelial cells.

The only observation on the innervation of blood vessels in the inner ear was made by Lorente de No,[15] who found nerve fibres passing from the cochlear nerve to the blood vessels in the modiolus of the rat. He could not determine the extent of these fibres but believed they were limited to the vessels in the modiolus. Contrary to his findings in the rat, numerous nerve fibres were found accompanying the labyrinthine artery in the internal auditory meatus in the guinea pig. They seemed to have no connection with the cochlear nerve. The nerve fibres were apparent only in relation to the vessels of the modiolus.

SUMMARY.

These studies were initiated with the purpose of filling in the details of capillary ramifications and relationships within the cochlea. By perfusion and special staining methods, the vascular pattern, the cytology of the vessel walls and innervation of the vessels have been studied in the guinea pig.

The capillary pattern in the spiral ligament has been described and the vessels of the region divided into four groups on a basis of course and location: one in the upper spiral ligament, one in the stria vascularis, one in the spiral prominence and a fourth in the depths of the spiral ligament.

The capillaries in the spiral lamina have been studied with an emphasis placed on their relationships and connections.

The capillaries of the stria vascularis appear to be composed only of endothelial cells. Smooth muscle cells were observed in the vessel walls of all other capillaries in the cochlea.

Nerve fibres were found to accompany the labyrinthine artery in the internal auditory meatus and to continue with the A. cochleae propria and its branches in the modiolus. No nerve fibres could be demonstrated with the capillaries of the spiral ligament or spiral lamina.

My sincere gratitude is expressed to the members of the Departments of Anatomy and Otolaryngology of Washington University School of Medicine, and the Research Staff of Central Institute for the Deaf for their constant interest and guidance.

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ACROMEGALY.

With Special Reference to the Otolaryngological Aspect.*

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INTRODUCTION AND MATERIALS

A recent report of the occurrence of bilateral abductor paralysis with acromegaly and the admission of such a case to the infirmary stimuated the review of 48 cases of acromegaly that were studied at the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital since 1935. These cases were reviewed with special attention paid to the otolaryngological picture. Passing comment is made of the general signs and symptoms, laboratory findings and treatment. This paper does not attempt to make a complete medical study of the disease. The findings should be of interest to the internist, the endocrinologist and the surgeon as well as to the otolaryngologist. They will point out the necessity of a complete otolaryngological examination in all cases of acromegaly.

To summarize the literature — in 1907, Neufeld published a bibliography of the work in this field up to that date; from 1907-1928, the literature is reviewed and summarized by Freystadtl; after 1928, five papers as listed in the bibliography have been published.

Signs and Symptoms:

The patients uniformly complain of general lassitude and weakness. Although there is the general increase in the size

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of the bones and a progressive coarsening of the features, the patients do not seem to notice it until the condition is far advanced. They may first notice that there is an increase in the hat or glove size. In patients with false dentures, failure of the plates to fit properly may be the first complaint. Malocclusion and inability to chew properly because of the marked prognathism may occur in patients with full dentures.

Otolaryngological:

Headaches occur in 70 per cent of the patients. The onset of the pain is in the occipital region with radiation to the frontal area. At first they are mild and fleeting, but with progression of the disease they become intractable. The pain is more severe in the morning and tends to diminish toward evening.

Face pain was present in 23 per cent of the patients. Only two of the cases showed causes other than the pituitary adenoma to account for the presence of the pain.

The distribution of the pain is over the maxillary division of the Vth nerve and in three cases was so severe that the nerve had to be sectioned for relief. Other cases were improved temporarily with alcohol injections of the nerve. Less often the face pain occurred over the ophthalmic distribution of the Vth nerve.

Another type of face pain may occur: this is the dull, gnawing pain that occurs over the jaw. It is explained by the rapid growth of the mandible with the resultant pressure on the nerves.

Laryngeal complaints were present in one-third of the patients. They varied from hoarseness and lowering of the voice to severe cough and dyspnea. There was one case of bilateral abductor paralysis in this series.

Difficulty in swallowing was present in five patients. Except for the generalized muscular weakness, no explanation could be found.

EXAMINATION.

There are many complete descriptions of the appearance of the far advanced changes in the acromegalic patient due to the effect of pituitary hormones which stimulate the growth of all tissues. There is a gross appearance of the face and typical acromegalic facies and habitus. The lower lip protrudes, the nose is large and broad, the expression is dull, and the supraorbital ridges are prominent. The teeth are short and often absent, due to the pressure of the bony growth. The tongue is characteristic. It is large, freely mobile, thick, and covered with coarse furrows. The mucous membrane of the oral cavity is hypertrophied and thick. The epiglottis is enlarged in all dimensions. The larynx, too, is enlarged, more in the lateral measurement, and the membranes are thick and redundant. Marie P. and Marinesco G. studied microscopical sections of these membranes in patients with acromegaly and reported that the changes found resemble those of pachydermia of the skin. The true cords are thick and white and move loosely with phonation.

Nasopharyngeal examination should be routine in these cases since the tumor may destroy the sphenoid and project into the nasopharynx. In the one case in this series where this was suspected the mass proved to be adenoid tissue.

It is in the examination of tne sinuses that the otolaryngologist may be the first to discover the presence of a pituitary tumor. In over 85 per cent of the cases, the sinuses were abnormally large and well aerated. Transillumination may cause this to be suspected. Examination of the visual fields showed a bitemporal hemianopsia in 30 per cent of the cases.

Laboratory — X-ray changes are present in 90 per cent of the cases. They vary from showing merely enlargement of the sinuses and extensive prognathism to complete destruction of the clinoids.

With the adenomatous growth of the pituitary gland, there are accompanying adenomatous growths in many other organs. Many tests to determine the presence of these growths have

been devised by the endocrinologists. A few of these studies are done periodically to determine the current status of the growth, to determine progression of the disease and to determine the effects of therapy. Some of the more routine studies are:

Serum phosphorus and phosphatase — Elevated.

17-Keto-steroids — Elevated.

B. M. R. — Elevated.

Blood sugar — Elevated.

Treatment:

Treatment of pituitary adenoma may be divided into three main groups — hormonal, X-ray and surgical.

Hormonal therapy was used in 15 per cent of the cases with some benefit. Recurrence and progression of symptoms was the rule and other treatment was necessary. ACTH was used in several cases, but the results thus far are inconclusive. Over 50 per cent of the cases were treated with X-ray. The average dose was 200 k.v. with a total dose of 1,000 r. through three portals, two lateral and one frontal. Two-thirds of the patients treated with X-ray were improved, but repeated courses of treatment were required. One patient received a total of 14,800 r.

The transphenoidal removal of the pituitary as done by Oscar Hirsch in selected cases offered the best chance of a complete remission of symptoms. Five cases done with this technique by Hirsch were improved. The neurosurgical approach, craniotomy, as done by the neurosurgical department of the M. G. H., gave good results in two cases.

Discussion:

Pituitary adenoma causes a large and varied group of clinical findings. The otolaryngological picture has been neglected because of the striking changes in the rest of the body. As pointed out by Grotting and Pemberton, if patients with acromegaly have a cord paralysis that is not found before operation it may endanger the successful outcome of the procedure. These authors reported five cases of bilateral vocal cord fixa-

tion (or paralysis) in acromegaly. Oscar Hirsch in a personal series of over 300 cases of acromegaly has not seen one case of bilateral abductor paralysis. The records of the infirmary do not mention any cases of cord paralysis except for the case reviewed. The cause of the bilateral vocal cord paralysis in this case may be explained by the presence of the mediastinal mass with pressure on the recurrent nerves. New and Childrey, in a review of 217 medical cases, found that malignant or benign enlargement of the thyroid may cause paralysis of the vocal cords. In their series there were 32 cases of cord paralysis from benign and 10 cases from malignant goiter. In two of the 42 cases, the paralysis was bilateral. Since the enlarged thyroids have been found to occur often in acromegaly, recurrent nerve pressure is probably the most logical explanation for the cord paralysis. It is definite that all patients with acromegaly should have a complete laryngological examination.

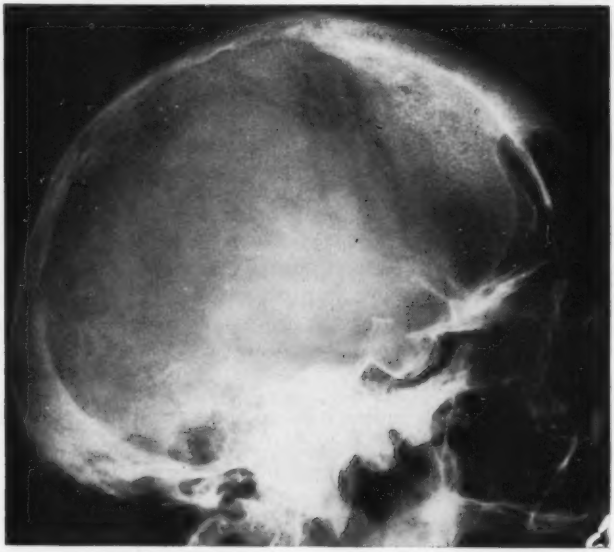

Fig. 1. Lateral view. X-ray showing typical skull findings in acromegaly: note abnormal enlargement of sinuses, thick cortex, ballooning of sella turcica, destruction of anterior and posterier clinoid proccesses.

CASE REPORT.*

A. J. 450115. The patient, a 73-year-old Russian and a former insurance agent, was admitted on March 19, 1951, with a chief complaint of respiratory difficulty for one year's duration with a marked increase in the severity of the symptoms for one week preceding admission. Over the past year, orthopnea, exertional dyspnea and ankle swelling had been present. Stridor, which was quite marked on admission, had been present for one week.

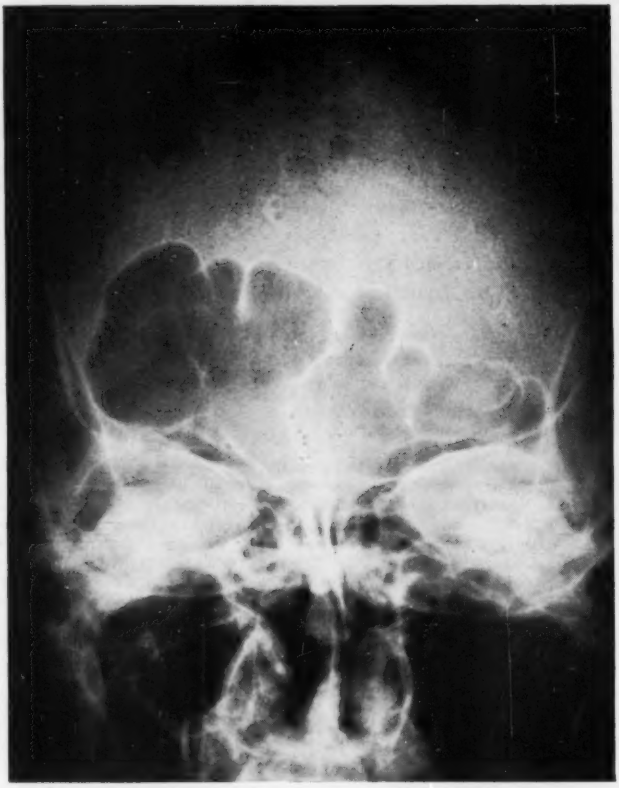

Fig. 2. A-P view.

*Presented at the meeting of the New England Otolaryngological Society on March 28, 1951. This case was included in a paper by Bauer and Aub (bibliography), in which particular attention was paid to the endocrinological aspect.

In 1944, the patient had presented himself at the throat clinic complaining of nasal obstruction, and a diagnosis of bilateral polypoid sinusitis was made. A routine laryngeal examination at that time revealed a left-sided abductor vocal cord paralysis for which no cause could be found. In 1946, both cords were paralyzed, but the patient had few complaints of respiratory difficulty. Until the current admission the patient was followed in the throat clinic. The advanced age of the patient prevented sinus surgery.

Past History: In 1926, the patient began to notice changes in the appearance of his face. His attention was first called to these changes by a friend who remarked that he looked like an old man and that his features were becoming coarse. In 1934, the patient noticed that his lower jaw was becoming more prominent and that his lower teeth overlapped his upper teeth. At about this time his voice became deeper. There were no complaints of headache, visual disturbance or personality changes. He later developed glaucoma which obscured any picture of temporal hemianopsia which may have occurred. The general physical condition of the patient, except for the marked weakness which accompanied his acromegaly, was fair. There had been complaints of orthopnea and exertional dyspnea since 1935. In 1936, a BMR was plus 26. On treatment with Lugol's solution it fell to plus two.

Previous Operations and Treatment:

1930—Excision of multiple fibropapillomata from neck and occipital region.

1935—Sialolithiasis with removal of the left submaxillary gland.

1936—X-ray treatment to pituitary adenoma with remission.

1944—Nasal polypectomy and removal of inferior turbinate.

1947—Chronic glaucoma.

1949—Removal of senile cataract.

Physical Examination: The patient showed typical acromegalic facies. Marked emphysema and kyphosis were present. Mild congestive heart failure was present with scattered rales over the bases of both lungs and a two plus pitting edema of the extremities. The nose was markedly enlarged. The naris on the left was filled with polyps and pus. The tongue was large, red and mobile, the larynx was large and the mucous membrane loose, thick and lusterless. The vocal cords were paralyzed in adduction with less than 1 mm. of airway. The excessive secretion and pus which had collected on the surface of the cords made the airway even smaller. The false cords were hypertrophied and the true cords were thick and white.

Laboratory Reports: Blood and urine within normal limits.

March 22, 1951—Serum calcium, 9.9. Phosphorus, 4.6. Phosphatase, 3.2.

March 20, 1951—Electrocardiogram — Auricular flutter, left ventricular hypertrophy.

Bacteriology—Nasal culture—Coagulase positive hemolytic staphylococcus aureus, E. coli.

X-ray:

March 19, 1951—Fluid in right base. Mass in superior mediastinum.

March 19, 1951—No obstruction to swallowing.

March 20, 1951—Skull bone thick and heavy, frontals large. Sinuses—Chronic purulent sinusitis, marked reaction around frontals and antrum.

In 1936, there was a report of large bones of hands, clubbing of phalanges, coarse trabeculation of bones. Sella enlarged and floor depressed.

Hospital Course: On admission, March 19, 1951, the patient was in severe respiratory distress, and a tracheotomy was done under local anesthesia. The patient was digitalized, and the cardiac congestion that was present on admission was relieved. Local treatment, including a shrinking spray and local irrigations was instituted in an attempt to improve the chronic sinusitis; chemotherapy was also given. The foul discharge gradually diminished and the nasal polyps have disappeared. Two days after admission the patient had difficulty in voiding and was transferred to the genitourinary service for treatment of an enlarged prostate. The patient withstood prostatectomy well. He has been discharged from the hospital and is being followed in the out-patient clinic. The tracheotomy tube is still in place and the patient has no complaints. It was concluded that the substernal mass probably represented an enlarged thyroid as it was present in films taken in 1936.

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MULTIPLE PAPILLOMA OF THE LARYNX:

A PRELIMINARY REPORT OF FOUR CASES TREATED WITH TERRAMYCIN.

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Papilloma of the larynx is the commonest laryngeal tumor of childhood. Although they are benign, the mortality rate may exceed that of carcinoma of the larynx in adults.[1]

Microscopically, these tumors are identical to the papillomas that occur in adults; however, they differ in several important aspects and should be considered as a separate clinical entity.

The incidence of cases has been reported as between one per 1,000 to one per 6,000 clinic patients.[2] The symptoms may occur from birth onwards; more commonly they develop before the age of five years. The sex incidence is about equal, and the racial incidence compares with that of the general population.

The etiology of the disease is unknown. Heredity and chronic iritation have been blamed, but there is little support for these theories. It has been repeatedly observed that papillomas in children tend to disappear at puberty, which suggests some hormonal influence; however, while the lesions tend to disappear in response to the hormonal changes of puberty, it does not necessarily follow that they occurred because of some hormonal influence or imbalance in childhood. Certainly, there are usually no other signs or symptoms to suggest such a disorder.

In 1923, Ullman[3] demonstrated what was presumed to be a virus in the laryngeal papillomas of a child, by two successive

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transfers of the lesion to the skin of adult volunteers. He also made three attempts to transfer a solitary papilloma of an adult to healthy skin, but without success.[4]

In 1933, Shope,[5] using material obtained from the skin papilloma of wild rabbits, was able to demonstrate that an agent which passed Berkefeld filters of V. N. or W. porosity was capable of producing similar lesions in the skin of other rabbits and was further able to demonstrate a neutralizing antibody to this agent in the infected animals. Rous,[6] using the Shope virus, was able to produce papillomas in the muscle and the viscera by direct implantation, and in the lungs by intravenous inoculation.

While not conclusive, it would seem that a virus plays some part in the production of papillomas, and perhaps this is altered by hormonal changes.

Grossly, the lesions appear as grayish-pink, mulberry-like nodules projecting from the mucosa, varying in size from a few millimeters in diameter to lesions that completely fill the larynx. They may occur anywhere in the larynx, trachea, bronchi, epiglottis or pharyngeal wall but are most commonly found on the true cords and ventricular bands. They are friable and bleed easily when touched.

Microscopically, they are seen to be benign, sessile or pedunculated lesions with a vascular connective tissue core covered by proliferated stratified squamous epithelium. They arise from the mucous membrane and show no tendency to infiltrate below the basement membrane. The cells show frequent mitotic figures indicating rapid growth; however, the cells are all well differentiated and mature.

The first symptom of papilloma of the larynx is usually a change in character of the voice, which is soon followed by a hoarseness which may ultimately result in aphonia. The first respiratory symptom is usually a cough. This is followed by progressive dyspnea which, if untreated, may result in asphyxia. A change in character of the child to an inactive, retiring type is not infrequent.[1] As the disease progresses. one

notes the increasing use of the accessory muscles of respiration, retraction of the supra- and infrasternal notches, and cyanosis.

Laryngeal papilloma in children is often a self-limited disease, the lesions disappearing at puberty. Smith[8] and Jackson[9] have reported cases that have undergone malignant degeneration, but all of these cases have been in adults.

If the papillomas fail to disappear at puberty, they usually take on the clinical characteristics of the adult type in that they show less tendency to recur and are often cured by simple surgical excision. A notable exception to this is a case reported by LeJeune,[10] in which the papilloma recurred in spite of 97 operative procedures and resulted fatally with a squamous cell carcinoma of the larynx.

The diagnosis is made by direct laryngoscopy, biopsy and microscopic examination of the tissue.

When confronted with a child who has chronic progressive hoarseness, one must, in addition to papilloma, consider other benign tumors, vocal nodules, "screamers' nodes," chronic laryngitis, congential cysts and webs, congenital laryngeal stridor, laryngotracheitis, hypertrophy of the ventricular bands, foreign body, paralysis of the recurrent laryngeal nerve, syphilis or tuberculosis.

The multiplicity of therapeutic procedures for multiple papilloma is evidence in itself of the failure of all of them to produce consistent results.

In regard to surgical procedures, it should always be borne in mind that in children the disease is often self-limited; therefore, nothing should be done that will result in any permanent deformity of the larynx. Gentle forceps removal under direct laryngoscopy is the most often practiced and is perhaps the best surgical procedure. This, at times, may be augmented by tracheotomy to obtain an adequate airway and may also be of value by placing the larynx at rest, as suggested by a case reported by Patterson,[11] in which recovery occurred following a tracheotomy only.

Numerous medications have been applied topically with varying and inconsistent success. Of interest is Broyles'[12] report of five cases treated by topical application of estrogens, amniotin in oil, in 1 cc. (10,000 U.) doses following surgical removal, in which a cure was obtained in all cases; however, Ferguson and Scott[2] were unable to duplicate these results with this therapy.

Varying results have been reported from the use of irradiation, both X-ray and radium. Of the two, external irradiation by X-rays seems preferable because of the lessened danger of perichondritis and the resultant permanent damage to the larynx;[13] however, with X-ray therapy the danger of producing permanent damage to the larynx still exists and is the chief contraindication to this method of therapy which, at times, seems to result in a cure.

Holinger[7] has recently given a preliminary report of seven cases of multiple papilloma in children treated with aureomycin in doses of 25 to 50 mgm. per kilogram per day following forceps removal. Two of the seven cases were apparent failures, but this may be the result of inadequate dosage of aureomycin. The remaining cases were all markedly improved. While it is yet too early to draw any definite conclusions, this form of therapy is rational and encouraging and deserves further trial.

The following is a report of four cases treated with terramycin* following forceps removal:

Case 1: F. J.,† a three-year-old colored male, who first came to our clinic on Sept. 6, 1950, with chief complaints of hoarseness and shortness of breath.

His illness apparently began when he was 19 months old, with an onset of hoarseness which gradually grew worse. In February, 1950, he was taken to another hospital in New Orleans because of this hoarseness, and a laryngoscopy was done, following which the child had more hoarseness and the mother refused to return with the child for further treatment. He was not seen again until he came to our clinic with a history of increasing respiratory distress of two weeks' duration and a marked increase in hoarseness.

*"Terrabon," elixir of terramycin, was used in the three children; 250 mgm. capsules of terramycin were used in the adult. Both were generously supplied by the Pfizer Co.

†From the service of Dr. Carl Granberry and Dr. Adrain Cairns at the Eye, Ear, Nose and Throat Hospital of New Orleans.

Past history revealed that he had frequent upper respiratory infections as an infant. He had measles at the age of two years.

Family history was negative for hoarseness, syphilis, tuberculosis or other pertinent findings.

Physical examination on admission revealed a child who could speak only in a whisper and who was in marked respiratory distress, using all the accessory muscles of respiration and revealing marked abdominal and suprasternal notch retraction on inspiration.

The patient was admitted to the hospital on Sept. 6, 1950, and placed in an oxygen tent. The following day, under local anesthesia, a low tracheotomy was done, the cannula being inserted between the third and fourth tracheal rings. Five days later, he was given an ether anesthetic and, under suspension laryngoscopy, it was seen that the entire larynx and supraglottic region was filled with papillomatous tissue. No visible airway was present. By gentle use of the forceps, the papilloma was removed as far as possible without injuring the cords. On the sixth post-operative day, he was started on terramycin, 50 mg. per kg. per day in four divided doses (one teaspoonful of terrabon every six hours). This was continued for 27 days, and four days after cessation of treatment he was re-examined under suspension laryngoscopy and the larynx was found to be perfectly normal.

On Dec. 5, 1950, six weeks after the previous examination, he was again examined under suspension laryngoscopy and there was seen to be a recurrence of papillomatous tissue which occupied approximately the anterior one-third of the glottic silhouette. This was gently removed by use of the forceps, and the patient was again put on terramycin in the same dosages as before and continued for 30 days.

On Jan. 16, 1951, he was again examined under suspension laryngoscopy and found to still have a small amount of papillomatous tissue at the anterior commissure. This was removed with a Mosher forceps. The terramycin was discontinued and 10 weeks later, on March 27, 1951, he was again examined with the suspension apparatus and multiple papilloma were removed from the right vocal cord in the posterior one-third and at the anterior commissure. At this writing, he has again been put on terramycin in a dosage of 250 mg. every six hours and will be kept on this for a minimum of two months.

Case 2: P. L. G.,* a six-year-old white female, whom I saw for the first time on Jan. 4, 1951.

Her chief complaint was hoarseness and shortness of breath. Her illness was noticed when she first learned to talk at the age of 18 months. Her voice was weak and she was able to speak only in a hoarse whisper. Her voice gradually became even worse and in February, 1947, two and one-half years after the onset, she began having progressively severe respiratory difficulty. On June 30, 1949, because of her respiratory difficulty, she was taken to another hospital where, under suspension laryngoscopy, multiple papilloma were removed from the larynx. Following this, she was apparently normal for two to three months, after which she again developed hoarseness and dyspnea, necessitating a second suspension laryngoscopy for removal of multiple papilloma. This was done on

*From the service of Dr. Kotz Allen and Dr. Mercer Lynch at the Eye, Ear, Nose and Throat Hospital, New Orleans, La.

Feb. 9, 1950. On March 5, 1950, May 5, 1950, July 19, 1950 and on Oct. 6, 1950, she had papilloma removed from her larynx under suspension laryngoscopy, all procedures being mandatory because of respiratory difficulty.

Past history and family history were essentially negative.

On Jan. 4, 1951, she was admitted to the Eye, Ear, Nose and Throat Hospital in marked respiratory distress. She was taken to the operating room for suspension laryngoscopy; however, during the induction of anesthesia with vinyl ether, she vomited and became markedly cyanotic. A tracheotomy was immediately done and the patient promptly responded. The anesthesia was then continued and the larynx exposed with the suspension apparatus revealing the entire glottic chink to be obscured by the papilloma. The papilloma were removed so far as was possible without damaging the vocal cords, and two days later she was started on terramycin, 250 mgm. per kg. per day (250 mgm. every six hours).

On Feb. 2, 1951, she was examined with the laryngeal mirror and found still to have some papillomatous growth. The terramycin was continued in the same dosage and on March 21, 1951, she was admitted to the hospital and her larynx examined under suspension laryngoscopy was found to be free of papilloma. Terramycin will be continued for at least another month, after which time she shall return for re-examination.

Case 3: T. J.,* a 28-year-old colored female, who in 1945 noted for the first time the gradual onset of hoarseness. The hoarseness was progressive; however, there was no dyspnea. On June 26, 1945, she had multiple papilloma removed from her larynx at another hospital. She was improved for about one week, following which the hoarsness became progressively worse, so that by August, 1945, she was unable to speak. On Aug. 7, 1945, she again had papilloma removed from her larynx. Following this, she was improved; however, her voice did not return to normal. In June, 1949, her voice again began to get progressively worse, but she received no treatment until she came to our clinic for the first time on Dec. 28, 1950, where examination revealed multiple papilloma on both cords and subglottically. On Jan. 10, 1951, under suspension laryngoscopy, the papilloma were removed with Mosher forceps and the following day she was started on terramycin in a dosage of 50 mgm. per kg. per day in four divided doses.

Past and family histories were essentially negative.

She has been followed at weekly intervals and one could note the gradual disappearance of the small amounts of papillomatous tissue that remained after surgery. On March 3, 1951, the larynx was free of papilloma, although the edges of the cords were still slightly irregular. This was attributed to the three previous procedures she has had on her larynx. At this writing, she is still on terramycin in the same dosage and will be continued on this for at least another month.

Case 4: P. D. C.,† a four and one-half-year-old white male, with the chief complaint of dyspnea and hoarseness.

*From the service of Dr. Kotz Allen and Dr. Mercer Lynch at the Eye, Ear, Nose and Throat Hospital of New Orleans, La.

†From the service of Dr. Kotz Allen and Dr. Mercer Lynch at the Eye, Ear, Nose and Throat Hospital of New Orleans, La.

His illness began at about the age of two years with the gradual onset of hoarseness, followed in several months by the development of respiratory difficulty which gradually became worse. On March 19, 1949, he was taken to another hospital where, under suspension laryngoscopy, multiple papilloma were removed from both cords, the arytenoids and the subglottic tissues. Following this, his voice began to get worse again, and in March, 1950, he again developed dyspnea, necessitating a second operation on March 10, 1950. On May 19, 1950, and again on Sept. 15, 1950, he required suspension laryngoscopy and removal of papilloma because of hoarseness and dyspnea.

Past and family histories revealed no significant findings.

On Jan. 3, 1951, he was admitted to the Eye, Ear, Nose and Throat Hospital where, under suspension laryngoscopy, multiple papilloma were removed from both vocal cords, the rest of the larynx appearing normal.

On Jan. 14, 1951, he was started on terramycin in a dosage of 50 mg. per kg. per day in four divided doses. Two months later, on March 16, 1951, he was readmitted to the hospital and the larynx examined under suspension laryngoscopy. He was found to have a small amount of papillomatous tissue at the anterior commissure, which was removed with Mosher forceps. The rest of the cords and the larynx were free of papilloma. At this writing, he is still on terramycin in the same dosage and will return for re-examination in one month.

In none of the cases was there any gastrointestinal disorders during treatment with terramycin. Complete blood counts were seen on all patients at the end of two months' therapy and all were within normal limits.

SUMMARY.

The incidence, etiology, pathology, symptomatology and treatment of multiple papilloma of the larynx are reviewed and discussed. Four cases of multiple papilloma of the larynx treated by surgery followed by terramycin are reported. Two cases are free of papilloma after two months of treatment. Two cases are improved but still have papilloma.

Complete blood counts on all the patients after two months of treatment with terramycin were within normal limits.

CONCLUSIONS.

Four cases are not enough on which to base any dogmatic statements, and not enough time has elapsed even to prophesy

what the eventual outcome will be in these four cases; however, I feel that there is sufficient evidence on which to base several general statements.

Terramycin in relatively large doses, given after surgery, appears to have some beneficial effects in the treatment of multiple papilloma. In a dosage of 50 mg./kg./day, it seems necessary to give it for a period of not less than two months. The apparent effect of terramycin on multiple papilloma lends added support to the theory of a virus etiology of multiple papilloma.

Terramycin in the dosage used is apparently nontoxic, there having been no gastrointestinal disorders or significant changes in the blood picture of any of the cases.

Case 2 well illustrates the danger of attempting to give a general anesthetic to an individual with respiratory obstruction. All patients with respiratory obstruction should first have a low tracheotomy done under local anesthesia. This was not done in Case 2 only because of the parents' objections to tracheotomy.

Case 3, although an adult, was included in this study because of its unusual similarity to the childhood form of multiple papilloma.

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OTITIS EXTERNA IN PUERTO RICO.*†

A CLINICAL AND BACTERIOLOGICAL STUDY OF 82 CASES.

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The infections of the external ear canal constitute a considerable part of the routine ear, nose and throat office practice. They have been estimated to comprise about 5 to 20 per cent of all such practice, depending upon the season of the year. While the condition is frequently seen in tropical and subtropical countries during all seasons, still, both in these countries and in the temperate zones, it is more prevalent during the Summer.

Many synonyms have been applied to this disease: otomycosis, fungus ear, mildew ear, Panama ear, Singapore ear, swimming ear, itching ear, adobe ear, Dhobie ear, jungle rot, stink ear, Hong Kong ear and hot weather ear. Clinically, it is mainly an illness of tropical and subtropical countries, characterized by itching, swelling, pain, a sensation of fullness in the ear, discharge, deafness and, at times, fever and trismus. It may be an acute process or a chronic condition of many months' duration. In the more acute cases, more of the above signs and symptoms are present at one and the same time.

Gill[1] has pointed out as predisposing factors the following conditions: maceration of the canal, traumatism due to foreign bodies, climatic influences and individual susceptibility.

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Gordon,[2] who made his observations in the island of Guam, thought that climatic conditions and a low level of personal hygiene were the most predisposing factors. According to Simonton,[3] atmospheric humidity was the primary factor in 341 cases studied in New Guinea and the Philippines. This author places cerumen as the ranking predisposing cause, and believes swimming and bathing to be of secondary importance only. Mac Cormack and Martin[4] believe that there may be a relationship between seborrheic dermatitis of the scalp and the otitis externa.

Until some 20 years ago the general opinion on the cause of this disease was that it was due to the action of fungi. This was primarily due to the influence of reports in the older literature, mostly from the tropics. Thus, Graham,[5] working in Syria in 1890, obtained mucor corymbifer from the auditory canal of a Moslem woman suffering from intense pain in the ear, itching and tinnitus; several forms of aspergilli were isolated at the same time.

In the more recent literature, some authors have continued to stress fungi as causative organisms. Simms[6] reported three cases in 1937 that he thought were due to aspergilli. Minchew, Collins and Harris[7] found, in 1940, no essential difference between the bacterial flora of the normal and the infected ear canal, but found 27 to 30 per cent of 93 suspected cases of otomycosis, to have aspergilli in their ears, while these fungi were relatively rare in normal ears. Barber *et al.*,[8] in 1931, reported pityrosporon as one of the commonest causes of the condition and thought that the staphylococcus aureus was responsible for the production of boils, and streptococci for fissures of the canal.

On the other hand, the opinion has been growing to the effect that the principal causative agents of otitis externa are bacteria, especially those of the pseudomonas aeruginosa (bacillus pyocyaneus) group and certain streptococci. In 1932, Hermitte[9] reported 56 cases of external otitis, refractory to all forms of treatment, in 21 of which he obtained pseudomonas, and brought about a cure by means of an autovaccine.

In 1934, Bettington[10] reported a cure of single case of pseudomonas otitis, also by autovaccine. Working in Egypt in 1938, Morley[11] reported 100 cases of the condition, in a certain number of which the cause again appears to have been pseudomonas aeruginosa. Other authors who found pseudomonas as the only or principal invader in cases of otitis were Greaves,[12] Dunlap,[13] Quayle,[14] Simon,[15] Beach and Hamilton,[16] Jones,[17] Senturia,[18] Syverton *et al.*,[19] Salvin and Lewis,[20] Clark[21] and Castle *et al.*[22]

Mitchell[23] and Ponce de León[24] stressed the importance of streptococci as pathogenic agents, an opinion that was much strengthened by the careful bacteriological work of Williams, Montgomery and Powell,[25] in 1939.

Other bacterial agents have been incriminated to a certain extent. For example, Salvin and Lewis[20] encountered, among 100 cases of external otitis, 27 with staphylococcus albus, 14 with various diphtheroids and nine with streptococcus viridans. Simon[15] isolated, in 90 patients, staphylococci from 20 per cent, bacillus proteus from 15 per cent, bacillus coli from 15 per cent, and diphtheroids from 6 per cent; however, in both of the two last mentioned studies, pseudomonas aeruginosa was the organism most frequently encountered in both pure and mixed infections.

In view of the importance of otitis externa in otorhinologic practice, of the fact that the opinion still prevails in certain sectors of the profession as to its fungous cause, and because of the disposal of sufficient and adequate clinical material for study, we decided to investigate the condition from the etiologic and therapeutic points of view.

METHOD OF STUDY.

A total of 82 out-patients in different stages of otitis externa, both acute and chronic, were selected. Material was taken from 112 ears, by carefully swabbing the external ear canal for use in the bacteriological studies, and then scraping

gently with sterile curettes to obtain material for the mycological work. These samples were immediately sent to the bacteriologist and mycologist, respectively.

The results of this study may be briefly and conveniently summarized as they appear in Table 1.

TABLE 1.

Organism	Pure Infections (No. of Cases)	Mixed and Pure Infections (No. of Cases)	Per Cent
Staphylococci	19	42	36.1
Ps. aeruginosa	9	21	18.1
Diphtheroids	7	23	19.8
Aspergilli	4	10	8.6
Streptococci	0	10	8.6
N. catarrhalis	3	5	4.3
B. coli	2	4	3.4
Penicillium	1	1	0.86

It is clearly seen that fungi play a negligible rôle, at least in the community in which the study was conducted, and that the most important organisms causing otitis externa are staphylococci, in the first place, and pseudomonas aeruginosa, in the second. The largest number of pure infections was 19, all of them due to staphylococci, which also predominated among the mixed infections. While pseudomonas was slightly more frequently encountered (nine cases) as a pure infection than the diphtheroids (seven cases), the total number of times that the latter were isolated was only slightly larger than that of pseudomonas; however, we attach no pathogenetic significance to the presence of diphtheroids in the external ear canal.

As regards mixed infections, we found that the largest number in which a certain organism appeared in combination with one or more was six cases, and these were staphylococci with diphtheroids and staphylococci with pseudomonas. It will be noted that both were examples of combinations with staphylococci, but since the total number of cases was only six for each combination of organisms, it is seen that this is not of statistical importance.

TREATMENT.

A large number of therapeutic agents have been used for the treatment of otitis externa. McBurney and Searcy,[26] in 1936, studied 69 agents that had been used in the past as fungicides, and found 2 per cent thymol in alcohol to be the best. Gill[1] and Whalen[27] recommend metacresyl acetate; Senturia[28-30] used sulfanilamide, penicillin and streptomycin; Goldstein,[31] 1 per cent thymol in cresatin; Brown and Kelemen,[32] glycerite of hydrogen peroxide; Wells,[33] penicillin in glycerin; Dean,[34] a powder compounded with penicillin, boric acid and powdered sulfathiazole; Spence,[35] 5 per cent sulfanilamide ointment, and McLaurin[36,37] uses sulfamylon and X-ray therapy in small doses.

In a comparative therapeutic study, Reech[38] found that mere cleansing of the external canal cured six of 11 patients seen in the dry season and seven of 15 treated during the wet season.

In the present study we adhered to a very simple form of treatment. The external ear canal was cleaned thoroughly by means of curettes and suction tips. Rarely was irrigation employed, but when considered necessary a mild antiseptic solution (boric acid, aqueous metaphen or bicarbonate) was utilized, followed by thorough drying, after which 95 per cent alcohol was instilled, in order to absorb any remaining moisture.

When the cultures were reported positive for staphylococci or other Gram positive cocci, sulfathiazole or sulfanilamide powder was insufflated. When the canal was found to be excoriated, tender and sore, or when furunculosis was present, cleansing was carried out as above, and a cotton wick impregnated with a bland ointment was inserted in the canal, as far as the tympanic membrane, whenever possible. The patient was instructed to apply dry heat to the external ear in the latter cases, and if fever was present, some sulfonamide was given by mouth, or penicillin by injection. After the complete resolution of the condition, the patients were in-

structed to avoid getting water or soap into the ear canal and to instill one or two drops of 1 per cent thymol in 70 per cent alcohol, once a day, after bathing, for four to six weeks.

A sharp difference in the mode of response was encountered between acute and chronic cases, and between those in which staphylococci were isolated, as compared with the pseudomonas group. To begin with, staphylococci were generally isolated from the acute, and pseudomonas (bacillus pyocyaneus), from the chronic.

The above detailed treatment yielded very satisfactory results in all the acute cases from which Gram positive cocci were isolated.

The chronic, and the acute ones due to pseudomonas (bacillus pyocyaneus), failed to respond. After trying systematically a large number of the preparations recommended by other authors, with fair to poor results, it was found that instillations of weak dilutions of acetic acid, up to 5 per cent, yielded as good results as anything else. Pseudomonas, in pure or mixed cultures, were isolated from all of the chronic cases.

The average duration of treatment for the Gram positive group was 12 days, and for the pseudomonas, from two to six or eight weeks.

DISCUSSION.

In the community in which the present study was conducted, the cases of otitis externa can be divided into main groups, according to whether bacteriological cultures of the external ear canal yield Gram positive cocci or pseudomonas aeruginosa. The former organisms are found mostly in acute infections and the latter in chronic ones.

Fungi appear to play a very minor rôle as etiologic agents of otitis externa. They were isolated from only seven of the 82 cases. Only one of these was a pure culture of penicillium. The other six were aspergilli, combined with staphylococci in four instances, with bacillus coli in one, and with bacillus proteus in another.

The staphylococci isolated were *albus* in 25 cases and *aureus* in an equal number. They gave rise to no clinically detectable differences.

The results of cultures were of great clinical significance both as to acuteness or chronicity, and as to the results of treatment, as discussed above in that section of the paper.

CONCLUSIONS.

1. A bacteriologic, mycologic and therapeutic study was conducted on 82 cases of otitis externa.
2. Fungi were found to be of very minor etiologic importance.
3. In most cases, staphylococci were isolated in pure or mixed cultures, and these were mostly acute infections.
4. In the second largest group, pseudomonas aeruginosa (bacillus pyocyaneus) was present, and these were all chronic.
5. Treatment by simple cleansing, with or without the instillation of sulfanilamide or sulfathiazole powder, is curative in most cases, within 12 days, when the cultures have yielded Gram positive organisms.
6. Simple cleansing of the ear canal, followed by the instillation of weak acetic acid solution, yields good results when pseudomonas is present in the cultures, but not in less than two to eight weeks.

ACKNOWLEDGMENTS.

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PEARLY TUMOR OF THE CEREBELLOPONTINE ANGLE. REPORT OF A CASE WITH REVERSIBLE DEAFNESS.

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Pearly tumors of the cerebellopontine angle comprise only about 6 per cent of all tumors occurring in this region. Because of their characteristic mother-of-pearl appearance, they have been called "pearly tumors." Also known as cholesteatomas and epidermoids, they are benign, encapsulated, congenital neoplasms which arise from aberrent epidermal inclusions.

Unlike other tumors of the cerebellopontine angle, symptoms and signs referable to the VIIIth cranial nerve are unusual; of 13 cases reported by Gonzalez Revilla,[1] 10 patients had normal hearing and nine normal labyrinths. Olivecrona[2] reported normal VIIIth nerves in five of seven patients with pearly tumors.

The commonest symptom produced by pearly tumors of the cerebellopontine angle is pain referable to the Vth cranial nerve. It most frequently involves the third division and may mimic tic douloureux.[3,4] According to Olivecroma,[2] the typical manifestations of these neoplasms are: *1.* typical tic pain beginning in the third division of the trigeminal nerve in a young person; *2.* normal Roentgenogram of the skull; *3.* normal results of neurologic examination; and *4.* dislocation of the cisterna pontis and cisterna ambiens in the encephalogram.

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The following case is of interest for two reasons: first, return of hearing following removal of a cerebellopontine angle tumor is rare,[5-7] and adequate preoperative and postoperative records of hearing and labyrinthine function are even rarer; second, it seems worthwhile to emphasize again that such a lesion can mimic tic douloureux. The explanation for disappearance of the pain for more than 10 months after alcoholic injection of the mandibular nerve is admittedly obscure.

REPORT OF CASE.

R. P. Y., a white man aged 28 years, was first seen by one of us (D. H. E.) on Oct. 31, 1949, because of pain in the right side of the face, progressive deafness and tinnitus on the right side, all of three years' duration. The pain, which was sharp, lancinating and of brief duration, was located at first in the upper gums and teeth, but later extended to the lower jaw. In the beginning, attacks of pain occurred many times daily, but later there were remissions for as long as six weeks.

A brief examination revealed no abnormalities except a slight decrease in hearing on the right to watch and voice tests. Audiometric and labyrinthine studies were not done.

The diagnosis of tic douloureux was made. Complete relief of pain was obtained after alcoholic injection of the mandibular division of the right Vth nerve on Nov. 9, 1949.

The patient had no further difficulty until Sept. 26, 1950, when he noted mild pain in the right upper teeth and beginning disappearance of the numbness. Because of this pain he returned to us Oct. 6, 1950. The pain in the second division of the Vth nerve on the right side became severe, the deafness on the right became progressively worse, and slight nystagmus on looking to the right developed.

A presumptive diagnosis of a lesion of the right cerebellopontine angle was made. Otorhinolaryngologic examination revealed total deafness on the right, mild nerve deafness on the left, dead labyrinth on the right (no response to 30 cc. ice water) and a normal left labyrinth. The blood glucose level was elevated and there were 15 cells per cmm. in the cerebrospinal fluid. A Roentgenogram of the skull showed no abnormalities.

On Nov. 10, 1950, unilateral suboccipital craniotomy revealed a pearly looking tumor in the right cerebellopontine angle. One centimeter of the VIIIth nerve was seen between the tumor and the internal auditory meatus. The tumor concealed the Vth cranial nerve and displaced the IXth, Xth and XIth nerves caudally. After bloodless piecemeal removal, the compressed and displaced pons and the greatly elongated and flattened Vth nerve came into full view.

The patient returned to work on the twenty-fourth postoperative day. Since operation there has been a progressive return of hearing (see Table 1) and labyrinthine function (see Table 2).

TABLE 1. AUDIOGRAM (DECIBEL LOSS).

Cycles/Sec.	256	512	1024	2048	4096
10-25-50	No hearing with masking				
11-20-50	60	60	60	60	60
6-30-51	30	20	20	10	30

TABLE 2. CALORIC TEST, RIGHT EAR.

10-27-50	No response to stimulation with 30 cc. ice water.
6-30-51	Normal response to stimulation with 5 cc. ice water.

SUMMARY.

A case of pearly tumor of the cerebellopontine angle simulating tic douloureux is reported. It is of particular interest because return of hearing followed removal of the tumor.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

November 1, 1951.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

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Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

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Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)
Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

Silvertone Model 103BM.
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Silvertone Model J-92.
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Silvertone Model M-35.
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Distributor: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

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Manufacturer: Telex, Inc., Minneapolis 1, Minn.

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Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube Model No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

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TABLE HEARING AIDS.

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Manufacturer: Sonotone Corp., Elmsford, N. Y.

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Meeting: Royal York Hotel, Toronto, Canada (mornings only), May 20-22, 1952.

MID-WINTER MEETINGS.

Eastern Section: Waldorf-Astoria, New York City, Jan. 11, 1952.
Council Meeting: Waldorf-Astoria, New York City, Jan. 12, 1952.
Southern Section: Academy of Medicine, Atlanta, Ga., Jan. 14, 1952.
Middle Section: Indianapolis Athletic Club, Indianapolis, Ind., Jan. 28, 1952.
Western Section: County Medical Bldg., Los Angeles, Calif., Jan. 19,20, 1952.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich
Vice-Chairman: Dr. J. M. Robison, 1304 Walker Ave., Houston 2, Tex.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Royal York Hotel, Toronto, Canada, May 13-16, 1952.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James M. Robb, Detroit, Mich.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. William B. Clark, 211 Loyola Ave., New Orleans, La.
Vice-President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

Meeting: Palmer House, Chicago, Ill., October, 1952.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Wm. H. Evans, 24 Wick Ave., Youngstown, Ohio.
President-Elect: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22, Pa.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

Pres.: Dr. José Gros.
Sect.: Dr. Pedro Hernandez Gonzalo, Calle 8, 358, Havana, Cuba.
Meeting: Third Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Havana, Cuba, January 20-24, 1952.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

Time and Place: Caracas, Venezuela, 1953.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Herman J. Moersch.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Alden H. Miller.
Secretary-Treasurer: Dr. Victor Goodhill.
Chairman of Section on Ophthalmology: Dr. Dennis V. Smith.
Secretary of Section on Ophthalmology: Dr. Carroll McCoy.
Chairman of Section on Otolaryngology: Dr. Howard P. House.
Secretary of Section on Otolaryngology: Dr. Edwin Scobee.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.
Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21, N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. G. M. Billings, Morganton, N. C.
Secretary and Treasurer: Dr. MacLean B. Leath, High Point, N. C.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Lewis F. Morrison, 490 Post St., San Francisco, Calif.
Secretary-Treasurer: Dr. Howard P. House, 1136 W. Sixth St., Los Angeles 17, Calif.
Meeting: Salt Lake City, Utah, 1952.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Courses annually the last two weeks in January at Los Angeles, Calif.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Thomas F. Furlong, Jr.
Vice-President: Dr. Harry P. Schenck.
Treasurer: Dr. William J. Hitschler.
Secretary: Dr. John J. O'Keefe.
Executive Committee: Dr. Valentine M. Miller, Dr. C. L. Jackson, Dr. George L. Whelan.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Francis LeJeune, Ochsner Clinic, New Orleans, La.
Vice-Chairman: Dr. V. R. Hurst, 315 N. Center St., Longview, Tex.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Garnett P. Morison, Charles Town, W. V.
First Vice-President: Dr. Charles T. St. Clair, Jr., Bluefield, W. Va.
Second Vice-President: Dr. Arthur C. Chandler, Charleston, W. Va.
Secretary: Dr. Melvin W. McGehee, 425 Eleventh St., Huntington 1, W. Va.
Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Directors: Dr. Eugene C. Hartman, Parkersburg, W. Va.; Dr. Ivan Fawcett, Wheeling, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Meredith Ostrom, Rock Island, Ill.
President-Elect: Dr. G. C. Otrich, Belleville, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Victor M. Noubleau.
Secretary: Dr. Héctor R. Silva.
1o. Vocal: Dr. Salvador Mixco Pinto.
2o. Vocal: Dr. Daniel Alfredo Alfaro.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. Fernando Casadesus.
Vice-Presidente: Dr. Luis Sufie Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Sec. de Actas: Dr. Juan Berini.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER— CAMPINAS.

President: Dr. Joao Penido Burnier.
First Secretary: Dr. Gabriel Porto.
Second Secretary: Dr. Roberto Barbosa.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. F. J. Monteiro Sales and Dr. Jose Martins Rocha.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. Alberto P. Haedo.
Vice-Presidente: Dr. V. R. Carri.
Secretario: Dr. Renoto Segre.
Pro-Secretario: Dr. Carlos A. Gutierrez.
Tesorero: Dr. J. M. Tato.
Pro-Tesorero: Dr. Norberto Von Soubiron.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Carlos A. Cleves C.
Vice-Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Elpidio Posada.
Tresoreo: Dr. Mario Arenas A.

Books Received

Books and monographs received are acknowledged in this column. This notice may be regarded as a return courtesy to the publisher or author. Reviews will be published later as the editors may elect.

Notice of each book or monograph, including title, name of author, publisher, pagination, price, etc., will be presented in these notices so that our readers may have all data at their disposal for further inquiry.

MOTOR PHONETICS. A Study of Speech Movements in Action. By R. H. Stetson, Ph.D., Oscillograph Laboratory, Oberlin College. Two hundred pages with index. Second Edition. Amsterdam: North Holland Publishing Co., 1951. Price $3.50 postpaid.

THE NEW WAY TO BETTER HEARING THROUGH HEARING RE-EDUCATION. By Victor L. Browd, M.D., with Introduction by Robert West, Ph.D. Two hundred twenty-six pages, illustrated with charts, graphs and eight pages of color plates. New York (16), N. Y.: Crown Publishers, 419 Fourth Avenue, 1951. Price $3.00.

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